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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 4 AUGUST, 1926 NUMBER 2

BACTEREMIA ASSOCIATED WITH ACUTE HEMOLYTIC STREPTOCOCCIC THROAT INFECTIONS *

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Throat infections due to the hemolytic streptococcus have assumed a rôle of vast importance in recent years, on account of the almost unlimited variety of complications that may result as secondary manifestations. These secondary manifestations (or complications) are often difficult to follow and to correlate. That a bacteremia is responsible for many, if not a majority, of these complications is becoming increasingly evident. This is of major importance to the otologist, as there has been a widespread belief that a bacteremia associated with a mastoiditis is almost pathognomonic of a lateral sinus thrombosis. This is substantiated by many able and careful observers. Libman [1] was unable to demonstrate a bacteremia in any case of otitic disease without complication. Reik,[2] in discussing Libman's paper, agreed with Libman's findings. This is in accord with Hays,[3] and Oppenheimer and others in this country and Leutert, Kabrak, Neurnberg and others of Europe. However, Libman states that he has reason to believe from his studies of blood (4,700 cases) infection of various kinds, that a bacteremia in children can arise more easily than in adults from uncomplicated otitis media or mastoid disease. In this connection it must be remembered that obtaining a positive blood culture is frequently a difficult matter. The reasons for failure are:

1. The organisms may be so infrequent that none are obtained in the quantity of blood withdrawn. This is probably the most frequent cause for failure in those cases of bacteremia in which a manifest "septicemia" is not evident.

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* Candidate's thesis for entrance to the American Laryngological, Rhinological and Otological Society.

1. Libman, Emanuel: On the Importance of Bacteremia in the Diagnosis of Sinus Thrombosis of Otitic Origin: A Clinical and Bacteriological Study, Bull. Johns Hopkins Hosp. **24**:388 (Dec.) 1913.

2. Reik, H. O.: Society Proceedings, Johns Hopkins Hosp. Medical Society, Bull. Johns Hopkins Hosp. **24**:388 (Dec.) 1913.

3. Hays recently cited a case of recovery by blood transfusion.

2. The bacteremia may be of the intermittent type and the culture taken during the intermittent stage. Many of the bacteremias of otitic or tonsillar origin are possibly of this type.

3. Improper technic or the wrong culture mediums may result in failure. Most bacteriologic investigators of the hemolytic streptococcus insist that the poured "shake" culture as used by Brown [4] is necessary for the best results.

4. A thrombus or clot may be infected only in the central portion, leaving a sterile outer zone (Libman). However, a thrombus is not a necessary vehicle for the bacteria to gain entrance into the blood stream. In acute throat infection, the living bacteria themselves probably gain entrance into the blood stream without the formation of a thrombus.

Whatever the cause of failure, we know that, in many cases, there is much clinical evidence to support a diagnosis of a bacteremia when repeated blood cultures are negative.

STREPTOCOCCUS HEMOLYTICUS

The hemolytic streptococci are spherical or slightly oval. They form definite, often quite long, chains. Occasionally they are seen in pairs or in short chains. Their hemolytic activity is caused by a hemolysin called streptolysin, which gives rise to an antibody (Besredka). In severe infections the hemolysis may occur in the vessels with the resulting hemoglobinemia (Davis).

There are many classifications of the streptococcic family. Most investigators have followed Schottmuller in discarding the old term *Streptococcus pyogenes* for the terms *Streptococcus hemolyticus, Streptococcus viridans* and *Streptococcus mucosus,* on the basis of their reactions and appearances on blood mediums.

Gordon [5] classifies the streptococci according to their ability to hemolyze blood and to ferment sugars, raffinose and mannite. He recognizes three main groups.

	Hemolysis	Raffinose	Mannite
1. *Streptococcus pyogenes* or *hemolyticus*	+	—	—
2. *Streptococcus salivarius* or *viridans*	—	+	—
3. *Streptococcus fecalis* or *enterococcus*	—	—	+

Holman [6] in promulgating his general classifications of the streptococcus family, found that the great majority of his hemolytic strains fell into one group but was able to differentiate seven variant groups in addition. Kinsella and Swift [7] found twenty-eight hemolytic strains

4. Brown: Monograph of Rock, Inst. Med. Res., no. 9, 1919.

5. Gordon, M. H.: Classification of Streptococci, J. State Med. **30**:432-435 (Oct.) 1922.

6. Holman, W. L.: Streptococci, J. M. Res. **34**:377 (July 16) 1916.

7. Kinsella, R. A., and Swift, H. F.: Classification of Hemolytic Streptococci, J. Exper. Med. **28**:169 (Aug.) 1918.

yielded such marked cross-fixation as to lead to the conclusion that all were closely related or practically identical. Birkhaug [8] differentiated three groups of the hemolytic streptococcus, a group causing erysipelas, a group causing scarlet fever and a large series of miscellaneous hemolytic streptococci producing a variety of pyogenic infections. Dochez, Avery and Lancefield [9] demonstrated four major groups of the hemolytic streptococci on a basis of the degree and character of hemolysis produced.

Most observers recognize the following groups:

1. Beta type (β) which produces a gray biconvex or disklike colony, surrounded by a complete and clearly defined zone of hemolysis 2 to 4 mm. in diameter, with no greenish or yellowish discoloration of the medium and on microscopic examination no intact cells remain in the hemolyzed zone about the colony.

2. Alpha prime type ($\acute{\alpha}$), which is similar to the beta type, but under the microscope a few corpuscles are seen to remain throughout the hemolyzed zone. These incompletely hemolyzed corpuscles are more numerous next to the colony.

3. Alpha type (α), which is a green producing variety with no hemolysis.

4. Gamma type (γ), which forms a gray color with no hemolysis.

Of the four groups the beta type is the one with which we are primarily concerned, as the other types are apparently not so virulently pathologic for man. Bloomfield and Felty,[10] in a study of 1,000 cultures of the hemolytic streptococcus were unable to recover from a case of tonsillitis, erysipelas, scarlet fever or other acute streptococcic infections, a strain which did not have typical and undoubted beta type characteristics. They insist the alpha prime, alpha and gamma types, are nonpathologic for man. The hemolytic activity of this organism is undoubtedly an indicator of the pathogenicity.

Dochez,[11] Gordon [12] and others have shown that the type of beta hemolytic streptococcus found in scarlatinal angina is, in general, a specific type distinguishable from the types of *Streptococcus hemolyticus*

8. Birkhaug, Konrad E.: Studies on the Biology of the Streptococcus Erysipelatis, Bull. Johns Hopkins Hosp. **37**:85-90 (Aug.) 1925.

9. Dochez, A. R.; Avery, O. T., and Lancefield, R. C.: Biology of Streptococcus, J. Exper. Med. **30**:179 (Sept.) 1919.

10. Bloomfield, A. L., and Felty, A. R.: On the Dissemination of Hemolytic Streptococci Among a Group of Healthy People, Bull. Johns Hopkins Hosp. **34**:414-417 (Dec.) 1923.

11. Dochez, A. R., and Sherman, L.: Significance of Streptococcus Hemolyticus in Scarlet Fever, J. A. M. A. **82**:542-544 (Feb. 16) 1924.

12. Gordon, M. H.: Serological Study of Hemolytic Streptococci, Brit. M. J. **1**:632 (April 30) 1921.

causing other kinds of angina and septic conditions in general. Bliss [13] has shown that this streptococcus scarlatina can be recovered from all patients with scarlet fever, as well as from contacts and healthy carriers.

Beckwith and Eddie [14] have shown that *Streptococcus hemolyticus* is very resistant to cold. They found it uninfluenced by freezing temperature, but readily killed by heat.

EPIDEMICS

In recent years, about a dozen or more epidemics of "septic" sore throats have been reported. Bacteriologic studies have invariably shown a streptococcus as the offending organism. Capps [15] described it as a capsulated hemolytic streptococcus. Davis and Rosenow [16] called it a peculiar streptococcus. Later, Davis acknowledged its relationship to *Streptococcus hemolyticus,* and thought that the two were identical.

A few of these epidemics were traced to infected milk. The Boston epidemic of 1911 was the first in this country to be so attributed. The investigations of Krumwiede and Valentine [17] and Davis and Capps [18] indicate that the animal is a secondary host, having been infected originally by a human being. The infection is carried through an abrasion of the teat or udder. Davis and Capps [18] have shown that when the hemolytic streptococcus is planted on the teat with an abrasion, the infection may ascend the ducts and cause a prolonged infection in the udder. The streptococci are shed in the milk in large numbers for weeks and months.

In winter months these epidemics of sore throat almost assume the proportion of a pandemic. The clinical picture of an acute throat (upper respiratory tract) infection is a mild coryza, with a sensation of fulness or dryness of the pharynx with slight discomfort on swallowing. Headache, frontal or occipital, may be present. Sense of malaise is rather marked and out of proportion to the appearance of the throat. Chilliness and slight fever is usually present, especially in children.

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13. Bliss, W. P.: Biology of Streptococcus, Antigenic Relationship Between Strains of Streptococcus Hemolyticus Isolated from Scarlet Fever, J. Exper. Med **36**:575-605 (Nov.) 1922.

14. Beckwith, T. D., and Eddie, B. U.: Hemolytic Streptococci of Human Origin in Relation to Heating and Freezing in Milk Industry, J. Lab. & Clin. Med. **9**:316-321 (Feb.) 1924.

15. Capps, J. A.: Epidemic of Sore Throat Due to Milk, J. A. M. A. **58**:1111 (April 13) 1912.

16. Davis, D. J., and Rosenow, E. C.: Epidemic of Sore Throat Due to a Peculiar Streptococcus, J. A. M. A. **58**:773 (March 16) 1912.

17. Krumwiede and Valentine: Coll. Stud. Bureau of Lab. City of N. Y., 1914-1915.

188. Davis, D. J., and Capps, J. A.: Experimental Bovine Mastitis Produced with Hemolytic Streptococcus of Human Origin, J. Infect. Dis. **15**:135 (July) 1914.

The temperature in uncomplicated cases lasts from three to four days. A secondary rise is not uncommon. The back and legs may ache, but just as frequently the aching is absent. Occasionally, a slight nausea and vomiting are present in children. Hoarseness is uncommon, but a slight tickling cough is usually present. The face is flushed, and occasionally an erythema over the chest is found. The anterior cervical lymph glands are usually tender and slightly swollen. The posterior lymph glands are occasionally involved. A mild conjunctivitis is present in almost 100 per cent of the cases during the acute stage.

The throat is diffusely reddened and inflamed. The uvula is edematous and hyperemic. The tongue is coated and the breath fetid. All the lymphatic structures of the mouth and pharynx are red and swollen. The tonsils may be spotted, but more frequently are only inflamed, similar to the pharynx.

A moderate leukocytosis of 15,000 to 18,000 is usual, persisting from four to eight days after the temperature has become normal. The red cells are not affected in uncomplicated cases. In those cases of acute otitis media or acute throat infection in which a bacteremia is suspected, the patient is usually bright eyed and alert (mental stimulation). There is no clouding of the intellect, even with a high temperature. Delirium is usually absent. The appetite is good, with a desire for much water. The temperature usually has a fall and rise during the twenty-four hours. The patient feels fairly comfortable. If it is an otitic case and an operation is performed, there is usually a drop in the temperature but a subsequent rise. The abscesses of a "septicemia" are comparatively rare in this type of a bacteremia.

Felty and Hodges,[19] in a carefully worked up series of forty cases of acute throat infection, report obtaining practically pure cultures of the beta hemolytic streptococcus in 100 per cent of their cases during the acute stage of the infection. They believe that all cases of acute tonsillitis (pharyngeal lymphoid tissue) are due to the beta hemolytic streptococcus, and the disease itself should be fixed on a firm etiologic basis with a definite entity such as erysipelas, rather than a clinical syndrome, which follows any bacterial infection of the pharynx. According to Felty and Hodges, as well as Bloomfield,[20] the green streptococci and gram-negative cocci represent the fixed normal flora of the mouth. This is not in accord with the usual conception of these organisms. Felty and Hodges attribute this supposed misconception to the employment of surface cultures, instead of the poured "shake" cultures as

19. Felty, A. R., and Hodges, A. B.: Clinical Study of Acute Streptococcus Infection of Pharyngeal Lymphoid Tissue, Bull. Johns Hopkins Hosp. **34**:330-335 (Oct.) 1923.

20. Bloomfield, A. L.: Significance of Bacteria Found in Throats of Healthy People, Bull. Johns Hopkins Hosp. **22**:33 (Feb.) 1921.

used by Brown.[4] Certain persons possess a natural immunity to this organism, as is true in other bacterial diseases. Most people, however, have repeated attacks.

Acquired immunity is of slight or short duration, as demonstrated clinically by the same person having the disease repeatedly and within a short space of time. Certain animals, such as the cow, mouse, cat and rabbit, are susceptible to the hemolytic streptococcus. Others, such as the rat, guinea-pig and birds, are immune.

CARRIERS

It may be conservatively estimated that during the winter months one half of the population of the temperate zone are active carriers of the hemolytic streptococcus. Pilot and Davis [21] recovered hemolytic streptococcus from surface swabs of the tonsils in 49 per cent of the patients examined. From the crypts they obtained the organism in 59 per cent of the cases, and from the crypts of the same tonsils after removal, they obtained a positive result in 92 per cent of the cases. The actinomyce-like granules in the tonsils show hemolytic streptococcus in large numbers and in a high percentage of instances (Pilot and Davis). Bloomfield and Felty [10] examined a group of 200 healthy women, and found about 41 per cent of them carriers of the beta hemolytic streptococcus. They determined such carriage was essentially dependent on focal tonsillar infection. It has been demonstrated that large outbreaks of epidemic meningitis may be preceded by a great rise in the carrier rate (Bloomfield and Felty).

Nakamura [22] examined over 2,000 extirpated tonsils, and found a striking increase in the incidence of the hemolytic streptococcus during the winter months, beginning in December and lasting until the middle of May.

Smillie [23] found that certain persons may retain the beta hemolytic streptococcus in the throat for three or four months after an attack of the disease. Sharp, Norton and Gordon [24] found a carrier condition of not less than eight weeks in five of eight instances.

In tonsillectomized throats, Van Dyke [25] found the hemolytic streptococcus in 16.4 per cent of the cases. This is about the percentage found

21. Pilot, I., and Davis, D. J.: Hemolytic Streptococci in Faucial Tonsils and Their Significance as Secondary Invaders, J. Infect. Dis. **24**:386 (April) 1910.

22. Nakamura, T.: Bacteriology of Extirpated Tonsils and Its Relation to Epidemic Tonsillitis, Ann. Surg. **79**:24-28 (Jan.) 1924.

23. Smillie, W. G.: Beta Hemolytic Streptococcus, J. Infect. Dis. **20**:45 (Jan.) 1917.

24. Sharp, W. B.; Norton, J. F., and Gordon, J. E.: Sore Throat Epidemic of Unusual Type: Influenza Studies, J. Infect. Dis. **30**:372-378 (April) 1922.

25. Van Dyke, H. B.: Hemolytic Streptococci in Normal Throats After Tonsillectomy, J. A. M. A. **74**:448 (Feb. 14) 1920.

by Nichols and Bryan [26] and by Pilot and Davis.[21] Tongs [27] found a somewhat lower percentage, and Simmons and Taylor [28] somewhat higher.

Pilot and Pearlman [29] examined the nasopharynx and adenoids of 103 children, aged from 5 to 16 years, with no evidence of a recent inflammation or sore throat, and recovered the organism from the nasopharynx and surface of the adenoids in 55 per cent of the cases. The crypts from the same adenoids produced the organism in 61 per cent of the children. The excised tonsils from the same children showed an incidence of 95 per cent.

Davis [30] found the organism not commonly present in the nose unless an active infection was present. He found the surface of the pharynx relatively free from the organisms in large numbers during the absence of the inflammation. In subjects with a cold, he obtained it in from 50 to 60 per cent of the cases. The normal gums do not harbor the infection in large numbers. They are rarely found on the skin if clean. They are found more frequently on the skin of people of unclean habits. Schachter [31] found hemolytic streptococcus in eight out of eighty-nine dirty people, usually from the hairy parts of the body. They are easily removed by washing. Their disappearance in the gastro-intestinal tract is equally rapid. When placed in the stomach of rabbits Davis [30] found the organisms dead in from two to five minutes. Valentine,[32] in his bacteriologic study of middle ear infections found, in the acute cases in which cultures were made at the time of the paracentesis and at intervals following, that the beta hemolytic streptococcus was the most important organism found and was recovered in nearly all cases. The bacterial flora changed after a few days.

The incidence of the hemolytic streptococcus in four common diseases by various observers gives an average as follows:

	Cases	Per Cent
Scarlet fever	337	85
Measles	1,245	48
Tonsillitis	128	71
Otitis media	204	74

26. Nichols, H. J., and Bryan, J. H.: Tonsil Infection, J. A. M. A. **71**:1813 (Nov. 30) 1918.

27. Tongs, M. S.: Hemolytic Streptococci in Nose and Throat, J. A. M. A. **73**:1050 (Oct. 4) 1919.

28. Simmons, J. S., and Taylor, A. E.: Bacterial Carriers in Upper Respiratory Tract, J. A. M. A. **72**:1885 (June 28) 1919.

29. Pilot, I., and Pearlman, S. J.: Bacteriological Studies of Upper Respiratory Passages, J. Infect. Dis. **29**:47 (July) 1921.

30. Davis, D. J.: Hemolytic Streptococcus with Special Reference to Their Rôle in Acute Respiratory Infections, J. A. M. A. **72**:319 (Feb. 1) 1919.

31. Schachter: Proc. Chicago Path. Soc. **10**:301, 1918.

32. Valentine, E.: Bacteriologic Study of Middle Ear Infections, J. Infect. Dis. **35**:177 (Aug.) 1924.

CHANNELS OF INFECTION

There are four main channels by which the infection may be transmitted to other portions of the body:

1. Spread by continuity, as evidenced by a peritonsillar abscess, infection traveling through the tissues of the neck and even invading the mediastinum, otitis media, mastoiditis, lateral sinus thrombosis, brain abscess, etc.

2. Entrance into the alimentary canal via the stomach. As before mentioned, Davis [30] has shown experimentally the fate of the hemolytic streptococcus when injected into the stomach of rabbits even in large numbers. They are all killed in from two to five minutes. Dible [33] only found two cases of hemolytic streptococcus in 359 stools. Goodby [34] examined the feces of fifty cases and failed to find *Streptococcus salivarius* (*viridans*) in any of them. Apparently the streptococci do not pass the barrier of the gastric juices in any appreciable number.

3. Extension by way of the lymphatics. This is demonstrated in many cases by a cervical adenitis, retropharyngeal abscess, etc. Goodman [35] and Mosher [36] report cases of primary jugular thrombosis secondary to a throat infection which they attributed to an extension by way of the lymphatics.

4. Direct infection of the blood stream. With the hemolytic streptococcus, it is highly probable that this is the most important route by which the bacteria are conveyed from the primary focus, whether of the acute or chronic type. A chronic bacteremia can most plausibly explain the term "subinfection," as used by Adami.

COMPLICATIONS BY WAY OF THE BLOOD STREAM

The possibility of a primary streptococcic peritonitis secondary to a throat infection by way of the blood stream has been denied until recently. The first positive case was that of Kunzler's [37] in 1904, in which the organism was recovered from the throat, peritoneal cavity and the blood. Necropsy revealed no intraperitoneal lesion. Somewhat later, Chapelle [38] reported eighteen cases of rapidly fatal peritonitis in children during an epidemic of sore throat. He demonstrated the organ-

33. Dible, J. H.: Enterococcus and Faecal Streptococci, J. Path. & Bacteriol. **24**:3 (Jan.) 1921.

34. Goodby, K. W.: Streptococcic Infections Arising from the Mouth, J. State Med. **30**:415 (Oct.) 1922.

35. Goodman, C.: Jugular Thrombosis, Ann. Otol., Rhinol. & Laryngol. **26**:527 (June) 1917; ibid. **24**:213-218, 1925.

36. Mosher, H. P.: Deep Cervical Abscess and Thrombosis of Internal Jugular Vein, Laryngoscope **30**:365 (June) 1920.

37. Kunzler: München. med. Wchnschr. **51**:1920, 1904.

38. Chapelle: Arb. a. d. path. Inst. zu Helsingfors **2**:583, 1907.

ism in the throat, peritoneal cavity and the blood. Rabinowitz [39] reports eight cases not in epidemic form, in which he established a preceding sore throat in most cases. The blood culture was positive thirty-six hours after the onset of the illness in one of the four cases in which the cultures were taken. Ransohoff and Greenebaum [40] report a case of acute hematogenous streptococcic peritonitis in a child, in which the same organism was recovered from the throat, peritoneal cavity and the blood. MacLennan and McNee [41] report a case of a child who had a primary hemolytic streptococcic peritonitis secondary to measles. Operation revealed a general peritonitis and a normal appendix. Pus from the abdomen and the blood culture showed the hemolytic streptococcus.

Poynton and Paine [42] showed that in young rabbits a micrococcus could produce a local lesion in the appendix by direct blood stream infection. Their later investigations [43] led them to believe that a cause of appendicitis may be a streptococcal invasion through the blood stream from a throat infection. They report a case that seems to prove conclusively that this is the case.

Rosenow's [44] experiments indicate that appendicitis frequently is a blood borne infection, secondary to some distant focus such as the throat. This would explain the occurrence of appendicitis, at times almost in epidemic form, when throat infections are quite prevalent.

Keegan [45] reports an epidemic of hemolytic streptococcic sore throat during February, 1919, at the U. S. Naval Hospital at Chelsea, Mass., in which attention was first called to the presence of an epidemic by the numerous instances of a postoperative rise in temperature in those who had abdominal surgical conditions. This rise in temperature was associated with a sore throat in all cases, and a subsequent infection of the previously clean wounds. Hemolytic streptococcus was recovered from the throat and the wound in all cases. Blood cultures were taken at various periods in ten cases, with negative results; however, the clinical evidence of a bacteremia in these cases could hardly be more strikingly

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39. Rabinowitz, M. A.: Acute Hematogenous Streptococcic Peritonitis, Am. J. M. Sc. **157**:797 (June) 1919.

40. Ransohoff, J. L., and Greenebaum, J. V.: Acute Appendicitis and Pneumonia in a Baby Fourteen Months of Age, Am. J. Dis. Child. **28**:76-78 (July) 1924.

41. MacLennan and McNee: Brit. J. Dis. Child. **10**:258, 1913.

42. Poynton, F. J., and Paine, A.: A Further Contribution to the Study of Rheumatism, Lancet **2**:1189-1191 (Oct. 28) 1911.

43. Poynton, F. J., and Paine, A.: The Etiology of Appendicitis as a Result of a Blood Infection, Lancet **2**: (Aug.) 1912.

44. Rosenow, E. C.: The Bacteriology of Appendicitis and Its Production by Intravenous Injection of Streptococci and Colon Bacilli, J. Infect. Dis. **16**:240-268 (March) 1915.

45. Keegan, J. J.: Hospital Epidemic of Streptococcic Sore Throat with Surgical Complications, J. A. M. A. **72**:1434 (May 17) 1919.

indicated than by the frequent deep infection of the traumatized tissue with the hemolytic streptococcus following a mild throat infection of the same organism.

Smith,[46] reports an epidemic of streptococcic hemolytic septicemia in a ward of seventeen babies, aged from 1 to 7½ months, secondary to a throat infection of the same organism. Seven babies died and came to necropsy. All gave a positive culture of *Streptococcus hemolyticus* from the heart's blood. Search of the literature reveals many occasions in which organisms have been recovered from the blood at necropsy, when repeated cultures had failed during life.

Bayne-Jones [47] reports an acute respiratory disease that suddenly broke out in twenty-five laboratory cats. The onset was sneezing, thin discharge from the nose and conjunctivitis. All but two cats died in fifteen days. At necropsy all the cats presented the same general appearance, a green mucopurulent exudate filled the cavities of the nose and extended over the nasopharynx to the bifurcation of the trachea. The cranial sinuses were normal. No evidence, gross or microscopic, could be found of involvement of other organs. The beta type hemolytic streptococcus was recovered from the throat and from plating a single drop of the heart's blood of these cats. Further experiments with this strain of bacteria showed an early loss of ability to produce the disease when placed within the nose of cats.

Brennemann [48] calls attention to the frequency of abdominal pain in children in throat infections, and suggests the probability of a blood borne infection to the mesenteric and retroperitoneal glands. He reports two such cases.

In many cases of arthritis, hemolytic streptococcus may be recovered from a gland removed from various portions of the body. Occasionally, blood from a vein contains such organisms (Crowe). This suggests that a bacteremia is not uncommon even in the absence of an acute infection.

Tanaka and Crowe [49] have shown by a series of carefully prepared slides that the epithelium that lines the crypts in a normal tonsil has a rich capillary blood supply. The large collecting veins that surround each crypt lie just under the basement membrane, and a destruction of this epithelium is the most common microscopic finding in chronic tonsillitis, thereby affording an easy pathway for entrance of bacteria into the blood stream. This entrance during an acute infection should

46. Smith, L. H.: Epidemic of Streptococcus Hemolyticus Septicemia, Am. J. Dis. Child. **24**:171-177 (Aug.) 1922.

47. Bayne-Jones, S.: Respiratory Infection and Septicemia of Cats Due to Hemolytic Streptococcus, J. Infect. Dis. **31**:474-479 (Nov.) 1922.

48. Brennemann, J.: Abdominal Pain of Throat Infections, Am. J. Dis. Child. **22**:493 (Nov.) 1922.

49. Tanaka, M., and Crowe, S. J.: Direct Blood Stream Infection Through Tonsils, Arch. Otolaryng. **1**:510-520 (May) 1925.

be doubly easy. The rather striking sequence of a cardiac involvement following an acute streptococcic throat infection is very suggestive of the possibilities of this method of disease transmission.

Acute thyroiditis is a frequent complication of hemolytic streptococcic throat infection. This is probably transmitted by way of the blood stream.

Fox and Hamburger,[50] in reporting a streptococcic epidemic following measles, note that it would seem more likely that the process was a primary septicemia, although the infection atrium was not clear in their cases.

Herrick [51] has shown that epidemic meningitis is preceded by a general invasion of the blood stream, and that in some cases the infection remains a bacteremia and never localizes in the meninges or elsewhere.

Warnekros,[52] in a series of twenty-five cases of intrapartum fever, demonstrated not only bacteria in the uterine secretions and in the placenta, but also a maternal bacteremia in 70 per cent of the cases. The infection of the mother was comparatively benign, only one patient died of infection.

McMeans [53] attempted to determine the effect of *Streptococcus hemolyticus* in the blood stream by injecting the organisms into the veins of rabbits, and found hemorrhagic lesions a constant feature. Necropsy demonstrated that the lungs, liver and spleen assumed an important rôle in combating this type of infection.

BACTEREMIA ASSOCIATED WITH ACUTE OTITIS MEDIA

In 1921, I [54] reported an epidemic of fifty-six cases (seventy-two ears) of an acute streptococcic otitis media of a distinct hemorrhagic character, secondary to an acute streptococcic throat infection. In almost every subject of this series there was a more or less septic temperature; many simulated the up and down temperature of a lateral sinus thrombosis. Following the paracentesis, which was performed in all these cases before rupture, there almost invariably occurred a rise in the temperature, in some cases to 105 F. This septic temperature persisted in a few patients for some weeks, and was uninfluenced by the free drainage obtained from an early and wide paracentesis.

The characteristic features of these cases were the persistent bloody serous discharge, later turning purulent, the fairly constant head pains;

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50. Fox, H., and Hamburger, W. W.: Streptococcus Epidemic at Camp Zachary Taylor, J. A. M. A. **70**:1758 (June 8) 1918.

51. Herrick, W. W.: Extrameningeal Meningococcus Infection, Arch. Int. Med. **23**:409 (April) 1919.

52. Warnekros: Plazentare Bakteriämie, Arch. f. Gynäk. **100**:173, 1913.

53. McMeans, J. W.: Experimental Bacteremia, Arch. Int. Med. **22**:114 (July) 1918.

54. Ballenger, H. C.: Acute Hemorrhagic Otitis Media, Ann. Otol., Rhinol. & Laryngol. **30**:539 (June) 1921.

the throbbing discomfort in the ears; the tender mastoids in all cases in the early stages; frequency of blisters on the eardrums, and the pumping action of the pus as it emerged through the incision of the membrana tympani. At the time this condition was reported, a bacteremia secondary to the otitis media was held responsible for the clinical course. In the only patient in whom a blood culture was taken, a streptococcus was recovered. In no case was a lateral sinus involvement demonstrated. All the patients recovered with dry ears. One of them required a simple mastoid operation for the persistent otorrhea. This occurred some weeks after the onset and after the stormy period had passed. At present, I would attribute greater importance to the throat infection as the etiologic factor of the evident bacteremia. The rise in temperature following the paracentisis could be attributed to a fresh invasion of the blood stream through the incised drum membrane.

Many cases of mastoidectomy performed in the first week of the otitis are reported in the literature. When the offending organism is the hemolytic streptococcus, an almost equal number of grave blood borne complications are also reported. It is logical to assume that, with this organism present, these complications, in the vast majority of cases, are accounted for by an entrance of the bacteria (not necessarily a thrombus) into the blood stream. The probability of this bacteremia is greatly enhanced by too early an interference, before the infected area is walled off by Nature's protective mechanism. Bacteriologic investigation as well as clinical observation tend to uphold this view. In these "too early" operations, the very complications we are trying to prevent are rendered thereby more certain of occurrence.

That these stormy cases of hemolytic streptococcic otitis do recover, in the vast majority of instances without surgical interference and without a lateral sinus thrombosis, is attested by my series of cases referred to in the foregoing. Harter,[55] Hays,[56] Maybaum,[57] Seydell,[58] Lake,[59] Dabney,[60] Webber [61] and others have reported cases of otitis media with a bacteremia, in which no demonstrable thrombosis of the sinus occurred.

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55. Harter, J. H.: Significance of Bacteremia Following Mastoid Operations, Northwest Med. **24**:35-37 (Jan.) 1925.

56. Hays, Harold: The Treatment of Sinus Thrombosis with Special Reference to Blood Transfusion, Eye, Ear, Nose & Throat Month., p. 447 (Oct.) 1923.

57. Maybaum, J. L.: Society Proceedings, New York Academy of Medicine, Laryngoscope **35**:557 (July) 1925.

58. Seydell, E. M.: Otitic Pyemia with Sinus Thrombosis, Ann. Otol., Rhinol. & Laryngol. **31**:862-868 (Sept.) 1922.

59. Lake, R.: Aural Bacteremia, J. Laryngol., Rhinol. & Otol. **34**:110 (April) 1919.

60. Dabney, V.: Bacteremia Due to Tonsillectomy Complicated by Mastoiditis, Arch. Otolaryng. **2**:362-364 (Oct.) 1925.

61. Webber, R.: Case of Bacteremia, J. Laryngol., Rhinol. & Otol. **34**:499 (Dec.) 1919.

CASE REPORTS

In going over the records of all cases of suspected bacteremia at the Evanston Hospital (courtesy of Dr. Williams) for the last nine years, it was found that 162 blood cultures were taken for various causes. Of these, 152, or about 94 per cent, were obtained during the last five years. In twenty-eight, a positive blood culture was found. Of these positive cases nine gave the cultural and agglutination reactions of *B. typhosus*. Of the remaining nineteen positive cases, a staphylococcus was found in eight instances, the pneumococcus in two, the green producing streptococci in three, an unclassified streptococcus in one and a gram-negative, nonpathologic organism in one. In only four cases was a hemolytic streptococcus demonstrated.

CASE 1.—Mrs. M. H. was operated on, April 19, 1922, by Dr. C. Danforth. A supravaginal hysterectomy and appendectomy were performed. Four days later a blood culture was taken, and many colonies of hemolytic streptococci were found. The preoperative examination revealed a lessened excursion of the left lower lobe but no other sign of involvement. The condition of the throat was not stated. Death occurred from peritonitis on the fourth day. The highest white count was 12,250.

CASE 2.—F. C. was seen by Dr. F. Christopher following a foot injury. The hemolytic streptococcus was obtained from the blood on the seventh day following the injury. The significant feature of this case was a sore throat following the injury but preceding the positive blood culture. The organism from the throat was not established.

CASE 3.—E. S., a boy, aged 3 years, reported by Dr. C. J. Swan.[62] Following a mastoid operation, numerous colonies of hemolytic streptococci were recovered from the blood stream. Opening the lateral sinus revealed no demonstrable thrombus. In view of the septicemia the jugular vein was ligated. The patient subsequently died of leptomeningitis.

CASE 4.—(This case is of more interest, and is an example of many similar cases seen by me.) H. R., a boy, aged 10, was admitted to the Evanston Hospital, Dec. 3, 1922. He gave a history of slight sore throat, nausea and vomiting beginning four days previous to his admission. The gastric upset lasted for a portion of the first day. The following two days he felt ill but was able to be up. On the third day (the day before admission) he had to go to bed with a temperature of 103 F. The morning of admission he had a temperature of 104.8 F.; redness of the conjunctival mucous membrane and a rash on the back were noted.

Physical Examination.—This revealed a well nourished, well developed boy of about 10 years. The skin of the face was flushed, with slight hyperemia elsewhere and a few patches of small red spots (pinhead size) on the back and in the axilla. The conjunctival mucous membrane was injected. The throat was moderately red and the tonsils were swollen with no patches. Koplik spots were absent. The eardrums were red but not changed otherwise. The cervical lymph nodes were not particularly enlarged. No rigidity of the neck or other signs of meningeal irritation were noted. The chest and abdomen were normal in every way. The extremities showed a slight cyanosis.

The following two days the temperature varied from 102.2 to 105 F., but his recovery was rapid from that time on. It reached normal on the fifth day of his admission, and remained there until his dismissal on the twenty-fifth day.

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62. Swan, C. J.: Ann. Otol., Rhinol. & Laryngol. **34**:872 (Sept.) 1925.

A blood culture taken on the second day of admission revealed hemolytic streptococcus. All other tests of the blood, urine and stools were normal, as were all other physical findings with the exceptions noted. The white blood count did not exceed 16,200.

CASE 5.—I. R., a girl, aged 12, was admitted to the Kenosha Hospital, Kenosha, Wis., Dec. 30, 1923, complaining of pain and a pus discharge from the right ear. One week before, acute rhinitis and a sore throat had developed, which was followed by a spontaneous rupture of the eardrum. The temperature, pain and tenderness had persisted, and on Jan. 1, 1924, a simple mastoidectomy was performed by Dr. T. Ashley of Kenosha, who found the usual mastoid findings of this stage, but with a normal appearing lateral sinus. The subsequent two weeks were stormy. The temperature had almost daily fluctuations from 98 to 105 F., accompanied by chills and profuse sweating. The white blood count did not exceed 10,000. Numerous cultures of the blood showed many colonies of the hemolytic streptococci. I saw the patient January 14, two weeks after the primary operation. In view of the very suggestive symptoms we reopened the mastoid, and found a normal appearing sinus. Incision of the sinus wall produced free bleeding from both ends. Nothing further was done other than to clean out a few remaining mastoid cells. The day following the second operation the temperature remained normal, but thereafter for another two weeks the septic temperature persisted, fluctuating from 97.6 to 104 F. From this time on the temperature gradually approached normal and remained so from February 5. The patient was discharged from hospital February 10 with dry ears and a healed wound. No metastatic abscesses or other evidence of a peripheral involvement developed. Her recovery was complete, with no apparent ill effects.

COMMENT

Case 3 demonstrates that a transient bacteremia of the hemolytic streptococcus is possible as secondary to a mild throat infection of the same organism. No physical findings could be demonstrated other than the bacteremia as a possible cause for the symptoms. The recovery was rapid and complete, with no apparent ill effects. If the temperature had not been so high or the rash had not appeared, the case would have resembled the thousands of other mild throat or "grip" cases, in which a bacteremia is not suspected and consequently a blood culture is not taken. It is possible that a transient or evanescent bacteremia is the rule rather than the exception in these mild throat infections of the hemolytic streptococcus. It would seem so from the clinical standpoint as well as from the reports of bacteriologic investigations that are obtained from the literature. The chances of demonstrating this bacteremia by the ordinary technic of blood cultures is not great, due to the difficulty, mentioned elsewhere in this paper, of obtaining a positive blood culture of this organism. In the event of traumatized tissue, as in the hospital epidemic reported by Keegan, the organisms found a fertile soil for development with the subsequent deep infection of the previously clean wounds. If these subjects had not been operated on, the epidemic probably would have been as other similar epidemics of sore throat, namely, a mild sore throat or a slight "grip" attack, with no suspicion of a bacteremia. Case 2 is suggestive of this. Cases 3 and 5 demon-

strate that a bacteremia of the hemolytic streptococcus in association with an acute mastoiditis is possible without a demonstrable thrombosis of the lateral sinus.

CONCLUSIONS

1. A bacteremia secondary to an acute hemolytic streptococcic throat infection is probably of common occurrence, and possibly is the usual accompaniment of this type of infection.

2. This bacteremia, if present, is of a transient or evanescent character in the vast majority of cases, and is not accompanied by the usual signs of a "septicemia" other than an elevation of temperature.

3. In a small percentage of instances, the hemolytic streptococcus lodges and multiplies in some distal portion of the body, and gives rise to the secondary manifestations characteristic of this type of throat infection.

4. With an acute otitis media or an acute mastoiditis present, a positive culture of the hemolytic streptococcus from the blood stream is not of as great diagnostic import, so far as a thrombosis of the lateral sinus is concerned, as are the other pus producing organisms.

HYSTERICAL DYSPHAGIA *

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AND
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Functional disturbances affect practically every organ in the human body, including the esophagus. They may manifest themselves in various forms, such as globus hystericus, and the symptoms may be multitudinous, but there is one type that stands separate from the other, to which H. S. Plummer, in 1914, gave the name of hysterical dysphagia. Hysterical dysphagia is characterized by a sense of obstruction at or about the esophageal introitus, is practically always associated with pallor and secondary anemia, and frequently with enlargement of the spleen. It must be remembered, however, that the line of demarcation between hysterical dysphagia and other types of functional dysphagia is not always clear or definite, although as a whole it represents a very definite clinical entity.

Vinson,[1] in 1922, published the first paper on the diagnosis and treatment of this disease. Since that time many more cases of dysphagia of a functional nature have been observed at the Mayo Clinic. In reviewing the entire group, we found sixty-five cases that fulfil all the requirements of true hysterical dysphagia. Many more that might be classified as such have been discarded on account of insufficient data. It is indeed interesting to note that all occurred in women, fifty-nine of whom were married. The average age for the entire group was 45 years, the youngest patient being 23, and the oldest 63. The average duration of symptoms was eight years, the shortest being one month and the longest thirty-four years.

SYMPTOMS

The dysphagia usually begins suddenly, in many instances the patient assigning the onset of her symptoms to some nervous shock when she strangled on a particle of food. The condition is usually as pronounced the first day of its appearance as at any time during its course. Some patients have periods when they are able to eat rather freely, then a sudden relapse to marked dysphagia. The relapse is practically always

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* From the Division of Medicine, Mayo Clinic.

* Read before the Section on Laryngology, Otology and Rhinology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

1. Vinson, P. P.: Hysterical Dysphagia, Minnesota Med. **5**:107-108, 1922.

instigated either by physical or mental fatigue. Solid foods, particularly meat and vegetables, are usually eliminated from the diet early. All solids are usually put through a grinder or are divided very finely in some other manner before deglutition. Liquids and soft foods, however, seldom cause difficulty. Pills and capsules, of all types and sizes, generally give the patients more trouble than any other substance. In spite of the restricted diet, there is seldom very little loss in weight, even when the disease has been present for many years.

Patients with hysterical dysphagia have a peculiar pallor which we now regard as one of its outstanding features. It is not the pallor of pernicious anemia although it may resemble it closely. A certain number of patients consult physicians because of the pallor rather than the dysphagia.

Of the sixty-five patients observed, only thirty-one had lost weight, the average loss being only 8 pounds (3.6 Kg.). No relationship was found to exist between the duration of the trouble and loss of weight. We are unable to say in what percentage pallor was present in the group under consideration, as there was no note made in several of the earlier cases, but in the last four years, we have found it to be present in every instance. In fifty-five cases the percentage of hemoglobin was 60 or less, the lowest being 27, while the average for the entire group was 48. The average erythrocyte count was 3,890,000; the average hemoglobin index was 0.6; the leukocyte count was 6,376, and in thirty-seven cases in which differential counts were made, they were normal. No relationship seemed to exist between the duration of the disease and the anemia.

The spleen was palpable in twenty cases, varying in size from slightly larger than normal to that of a tumor filling one-quarter of the abdomen. In one case in which the spleen was very large and the anemia very pronounced, the dysphagia was disregarded, and the condition was suspected to be splenic anemia although the blood findings were not typical. Splenectomy was considered, but because of the history of dysphagia the patient was treated for hysterical dysphagia, and under this simple procedure, the anemia improved and the spleen decreased in size. The duration and severity of the symptoms, as well as the degree of anemia, apparently had no direct relationship to the palpability of the spleen. The average hemoglobin percentage for the group with palpable spleens was 46, as compared with 48 for the group as a whole. The absence of teeth was so common that it deserves mention. Most of the patients had had their teeth extracted because of the dysphagia but without benefit, whereas others developed the dysphagia simultaneously with the extraction of the teeth or the use of new plates. The mucous membranes of the nasopharynx and mouth were often atrophic and dry, and cracks in the corner of the mouth were common.

Changes in the tongue were noted in eleven of the sixty-five cases. In some cases they closely resembled those found in cases of pernicious anemia. It is likely that, if the tongue had been carefully scrutinized, it would have shown changes in a much higher percentage. Test meals were given in ten cases, and achlorhydria was revealed in eight. Paresthesia was mentioned in five cases and diarrhea in five. It is probable that these symptoms had existed in a higher percentage without special attention being called to them.

During the last eighteen months a more detailed study of the character of the anemia in these cases has been undertaken. On esophagoscopic examination, the mucous membrane of the upper part of the esophagus was dry and atrophic, with loss of elasticity, and bled easily on manipulation. We have never been able to demonstrate the presence of an organic lesion, esophageal membrane or webs.

While roentgenologic examination of the introitus is notoriously difficult and mistakes are frequent, such examinations were attempted in every case. In fifty cases, the report was negative, eight patients would not swallow the barium mixture for fear of choking, three showed a slight narrowing high in the esophagus, three a slight lagging at the cardia, and in one the report was indeterminate.

COMMENT

Although the anemia is apparently of the secondary type with a low hemoglobin index, the rather frequent occurrence of changes in the tongue, much resembling those common in pernicious anemia, and the frequency of achlorhydria make one wonder if there may not be some relationship between the anemia of hysterical dysphagia and that of pernicious anemia. It seems quite likely that the anemia and achlorhydria result from the dietary restriction, especially with regard to meat, which in turn results from the dysphagia. On the other hand, one must consider the possibility that the dysphagia results in some cases from irritation of the esophageal introitus, accompanying esophagitis associated with the glossitis and stomatitis which accompany preexisting anemia.

Because the anemia resembles pernicious anemia in some of its symptoms, and because it seems most likely to be the result of dietary insufficiency, one must give some consideration to the interesting possibility that pernicious anemia may be dietary in origin. This possibility is enhanced by the fact that sprue and pellagra, which so closely resemble pernicious anemia at times, are considered by most observers to be deficiency diseases. The enlargement of the spleen in a high percentage of cases is another feature somewhat suggestive of a relationship between the anemia of hysterical dysphagia and pernicious anemia. However,

we believe that the anemia in these cases is not of the pernicious type, but that it is possible the anemia associated with this condition and pernicious anemia may have a common fundamental deficiency factor.

TREATMENT

The treatment consists in passing a plain esophageal sound into the stomach, guided by a previously swallowed silk thread. The size of the sound is immaterial. In those cases in which a slight spasm is reported at the cardia, the sound is simply passed through the introitus and not through the cardia. There is generally a slight amount of bleeding after passage of the sound, due to trauma. The treatment is purely suggestive, as the esophagus is not stretched. In the normal esophagus there is a narrowing at the introitus, and on passing a sound a slight obstruction is met at this point. It is a common mistake for physicians, after passing a sound, to report this normal narrowing as a stricture. Patients are then assured that they are now able to eat everything, and are encouraged especially to eat solid foods, meats and vegetables, and pills of ferrous carbonate are recommended. It is well worth while to learn the patient's social history so as to be able to point out to the patient the influence of psychic factors. With the explanation of these factors, our results have been better and more enduring. Usually nothing further is required to effect a cure. The dysphagia disappears, the blood picture improves at once, and the spleen may return to normal size. The disease may and does recur, especially if the patients have not been properly reassured. The most common cause for recurrence is some nervous shock or undue nervous strain. Recurrence generally develops in women who live under unfavorable conditions, and are very subject to psychic influences. In several, a recurrence took place after excessive menorrhagia or some unrelated disease. A second passage of the sound will always afford relief in such cases.

RESULTS

Prolonged observation has revealed most gratifying results. Replies were received to twenty-three questionnaires that were sent out. The average duration of time elapsing since treatment was three years. In this group the average hemoglobin at the time of treatment was 41 per cent, while the average for the same group after treatment for an average period of three years was 70 per cent, and practically all of the patients were cured, although most of them had intervals of slight recurrence when tired or under nervous tension. The results obtained within the last few years have not been tabulated because we felt that the time elapsed was not sufficient to afford an accurate conception of the results. In most instances we requested the patients to report in six months or a year for reexamination, but frequently they merely write that they are

feeling stronger and better and having very little trouble; therefore they see no necessity for reexamination.

ETIOLOGY

Hysterical dysphagia we regard as a functional disturbance primarily involving deglutition. By reviewing the history critically and especially by investigating the social aspect with care, one can always elicit the fact that the patient has been under undue nervous or physical strain and that the first symptoms occurred with some psychic upset. It is not uncommon for a patient to say that she had developed the syndrome as a defense mechanism against some unpleasant environment, and that it passed beyond her control. Another factor that adds weight to the hysterical theory is that in many cases the passage of stomach tubes and even sounds and esophagoscopes has not afforded relief, whereas later, with reassurance, the passage of a smaller sized instrument has given complete relief.

The question may arise whether the secondary anemia or the dysphagia occurs first. While the greatest number of patients seem to develop the anemia after dysphagia, we have not been able to check this point absolutely. Some patients give a history of pallor preceding the dysphagia, and others have received treatment for anemia previous to dysphagia.

Jacobson,[2] in reporting roentgenologic observations of the esophagus in patients with globus hystericus, considered the feeling of a lump in the throat due to a spasm of the esophagus. While none of our patients complained of a sensation of a lump in the throat, they said that during the periods when they were able to eat they could feel something relax in the throat. Might it not be possible that, due to some nervous strain, such a spasm occurs or is even brought on through an infection? In the final analysis, the condition may be found to be on the same basis as cardiospasm, the etiology of which is still indeterminate, with as many theories as investigators.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis is indeed interesting. All lesions producing dysphagia must be considered. Among the most common diseases that may produce dysphagia with a feeling of obstruction high in the neck are: malignant neoplasm, cicatricial stricture, diverticulum, bulbar palsy, cardiospasm, tuberculosis, syphilis, extraesophageal tumors pressing on the esophagus and congenital webs of the esophagus which are described in the literature. Hysterical dysphagia may be differentiated

2. Jacobson, Edmund: The Physiology of Globus Hystericus, J. A. M. A. **83**:911-913 (Sept. 20) 1924.

from these lesions by the following criteria: (1) Duration of symptoms, the average duration being eight years; (2) existence of anemia without loss of weight; (3) symptoms as marked on the first day as during the entire course of the disease, although deglutition may be practically normal at times; (4) usually ease in swallowing liquids and soft foods; (5) absence of clinical or physical evidence of tuberculosis or syphilis; (6) anamnesis negative for ingestion of corrosive substances; (7) enlargement of the spleen in more than 30 per cent of cases; (8) negative roentgenologic, Wassermann and neurologic examinations, and (9) absence of palpable or demonstrable extra-esophageal tumor causing pressure on the esophagus.

CONCLUSIONS

1. There exists a definite group of cases of dysphagia, characterized by secondary anemia and frequently by enlargement of the spleen with no demonstrable lesion in the esophagus, that we have termed hysterical dysphagia.

2. The disease is most predominant among women and is generally of long duration before diagnosis is made.

3. Loss of weight is rather uncommon in spite of restriction of solid foods, especially meats and vegetables.

4. The secondary anemia and enlargement of the spleen probably arise from a prolonged unbalanced diet.

5. Normal deglutition can be restored by passing an esophageal sound, and when this occurs, the blood picture usually returns to normal and the splenic enlargement may subside.

6. Recurrences are likely to occur, but relief can be afforded by further passage of sound and by constant reassurance of the patient.

DISCUSSION

Dr. John B. Potts, Omaha: I have observed the symptoms of hysterical dysphagia described by Dr. Moersch, but neither of the internists with whom I am associated have called my attention to it as an entity. The condition has been considered by me as an extreme condition of globus hystericus. The paper brings out the fact that all these people recover from the physical symptoms as well as the mental symptoms if they are fed properly, which would seem to indicate that the condition probably is due to insufficient food. Globus hystericus, in my opinion, covers a very large range of symptoms, anything from dryness of the mouth to a sinking sensation in the pit of the stomach, and the different manifestations in the throat and esophagus are a reflex from some emotion or an insult to their conscience or self respect. This emotion is usually due to early religious or moral training and hence the reaction to any stimulation or insult to the inner self differs in every person. If this individual act, thought, or whatever the stimulation may be, is of such a nature that it can be brought out, so to speak, in the open, no permanent injury to our subconscious self results. If, on the other hand, the individual act, thought, etc., is of such a nature that the natural reaction is to suppress it, then we have an insult to our subconscious self, which may or may not

do permanent injury. For this reason most of these stimuli have a sex basis. This leads to all degrees of mental depression, and this depression plus the globus with the resultant low feeding, produces the extreme condition which Dr. Moersch describes as hysterical dysphagia. In trying out this theory, for several years I have not passed a bougie or stomach tube on these globus patients, but instead have sent them for analysis to a competent neurologist. We have found that they could be relieved by proper analysis without any instrumentation.

Dr. Dan Brannin, Dallas, Texas: Hysterical dysphagia, like so-called "cardiospasm," is a beautifully clear and satisfactory entity so long as you treat it from an inferential deductive point of view; when, however, the patients are examined by the esophagoscope, many of them are found to have organic diseases of various kinds, ranging from a mild and easily curable esophagitis to cancer. When someone shall have subjected the question of supposed hysterical dysphagia to the same kind of study as that which Mosher has given the so-called "cardiospasm," a parallel revelation of an organic basis for a supposed purely functional condition will be demonstrated. By this it is not meant that there is no such condition as hysterical dysphagia; but rather that, like idiopathic meningitis, the cases will become fewer in number as our knowledge increases. At the Chevalier Jackson Clinic they have all been surprised at the number of times the preliminary, tentative, inferential diagnosis of hysterical dysphagia has had to be revised after performing the esophagoscopy that modern practice demands shall be done before such a diagnosis is allowed to stand. Over and over again they have found a lesion in these cases. In some instances the lesion has been secondary to functional disturbance of the deglutitory cycle, but nearly always the lesion has been found to be the primary cause of the functional difficulty, or of the symptoms. It is interesting to note that the histories of hundreds of cases of cancer of the esophagus at these clinics show that almost every case was at one stage inferentially and erroneously diagnosed hysterical dysphagia. So long as a diagnosis of esophageal disease is made inferentially, just so long will there be no chance for the surgeon or anyone else to cure cancer of the esophagus. Dr. Tucker's suggestion is that the author add to his paper that the esophagus should not be treated without looking at the esophagus, and that the diagnosis of hysterical dysphagia should never be made until the twenty or more organic diseases of the esophagus have been excluded by the simple method of looking at the esophagus with an esophagoscope.

Dr. E. Lee Myers, St. Louis: Dr. Moersch has helped me make a diagnosis more certain after many years. I have a case in mind—one of typical globus hystericus. An esophagoscope was used two or three times with practically no pathologic changes found. The patient's tonsils were removed, the lingual tonsil was cauterized and still there was no improvement in her condition. In examining her psychic condition, it was found that she had been treated poorly at the hands of her husband, who left her with four small children, and, consequently, she had lost her emotional balance. She was inclined to be a self-pitier. In this particular case an internist gave her pills of ferrous carbonate, and after her teeth had been taken out she was much better. However, no record was made of her blood counts and it is difficult to say what this condition would have shown; she has made a very good recovery. I agree with Dr. Potts who said that these cases have had some insult to their alter ego. Unquestionably, these people live in a world by themselves; some with a high personality complex, some with a low personality complex. They go around looking for sympathy and usually they are worse after consulting with a physician, who at the best has not studied their case physically.

Dr. C. W. Richardson, Washington, D. C.: To my mind Dr. Moersch struck the keynote, but he did not keep his finger on it. I have seen probably a dozen of these cases. I have seen them in young children, and I believe they are all

deficiency feeding cases. That is the point Dr. Moersch did not keep his finger on sufficiently long. He mentions that the condition could not be corrected except by suggestion, and then giving the patients proper food. I find that the same course is necessary in all of them; after a thorough diagnosis is made, use suggestion; but the most important thing is to put them on a balanced diet and give them pills of ferrous carbonate.

Dr. Herman J. Moersch, Rochester, Minn.: I believe that the points brought up by Dr. Tucker, as being essential before making a diagnosis of hysterical dysphagia, are covered completely in our paper. With reference to the possibility of confusing these cases with early cancer of the esophagus, it is only necessary to recall that the average duration of trouble was eight years before an examination, and the condition was as bad the first day of the disease as at any time during its course. While we feel that hysterical dysphagia is on a functional basis, we cannot be dogmatic on this point, as there may be a possibility of a preexisting anemia underlying the dysphagia. Necessarily, any further investigation or suggestions that will tend to clarify this point will be of great value. Dr. Potts laid emphasis on the etiology underlying globus hystericus and hysterical dysphagia. While they both may have the same etiologic basis, it is necessary to recall that none of our patients complained of a sense of obstruction in the throat.

CULTURES FROM TONSILS*

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During the past fifteen years, the faucial region of almost every person presenting himself to a laryngologist has been carefully scrutinized to ascertain clinically whether there is any local justification for the removal of the tonsils. The presence of pus in the follicles and substance of the tonsils is frequently described as the local evidence of the necessity for removal. The presence of pus, free pus, in these organs is an exceedingly rare condition from my observation; probably patients and physicians refer to inspissated secretion contained in the follicles. For some time I have been faced with the assertion, and no doubt many of the members of this association have had similar experience, that the tonsils must be cultured to determine the necessity for excision, the presence of streptococcus, staphylococcus or pneumococcus, and possibly of other cocci, being the determining factor. It has always been my firm conviction that the faucial region was one of the richest harbingers of bacilli; that it possessed a rich flora of various micro-organisms. This conviction has been intensified by numerous cultures made at various times during my professional career. The presence of bacteria is not inimical to well-being. It is the gaining of access into the organism and fructifying that creates ill health. In order to ascertain the value of the mere presence of bacteria in cultures from the tonsils as of any diagnostic significance, I determined to make a bacteriologic study of the subject. In this study, cultures were made in the usual way. A smear was made on the tonsil in situ while pressure was exerted to gain free milking from the follicles. Tonsils which were removed were smeared on the faucial surface, while compression of the tonsil was employed at the same time. In the tonsil fossae a vigorous rubbing was employed with the culture smear. In this study I proposed a bacteriologic test of three types of cases, namely: (1) faucial cavities from which tonsils had been removed, leaving a thoroughly clean fossa without adventitious tissue of any kind; (2) faucial regions wherein the tonsils did not present clinical evidence, general or local, requiring tonsillectomy; (3) faucial regions wherein the tonsils evidenced both distinct local and general clinical manifestations demanding their removal.

In the first type of cases only fossae which had aged and presented as perfect a result as it is possible to attain were smeared for culture. Fossae which presented any new growth of any kind were not employed.

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* Read at the American Laryngological Association, Montreal, Canada, June 4, 1926.

In the second type the smears for culture were made on persons who presented practically no tonsil or only a very small gland which had practically involuted, and without evidence of follicular infection. All of these patients were over 50 years of age.

In the third division, there were not only many very large tonsils, all of which showed evidence of chronic follicular inflammation, but also marked constitutional disturbances. The constitutional evidences were largely of some form of toxanemia.

It was my original intention to make several hundred cultures of each of the three groups, but as the findings were identical for each individual group, and so positive for the object in view, I desisted

Bacilli Found in Tonsils of Three Groups of Subjects

No. 1		No. 2		No. 3	
Staphylococcus	19	Staphylococcus	16	Staphylococcus	17
Streptococcus	9	Streptococcus	11	Streptococcus	9
Pneumococcus	6	Fusiform bacilli	1	Pneumococcus	9
Staphylococcus, streptococcus	4	Hay bacillus	3	Staphylococcus, streptococcus	3
Staphylococcus, pneumococcus	4	Sarsina	1	Hay bacillus	1
Hay bacillus	9	Staphylococcus, hay bacilli	1	Micrococcus catarrhalis	1
Micrococcus catarrhalis	2	Pneumococcus	1	Hemolytic streptococci	1
Pneumococcus catarrhalis	1	Pseudo-Klebs-Loeffler	2	Streptococcus, B. subtilis	1
Pseudo-Klebs-Loeffler bacillus	1	Micrococcus catarrhalis	1	Small gram-negative bacilli	1
				Streptococci, micrococci	4
				Mass. gram-negative micrococci, bacilli	2

after making over a hundred cultures. The bacteriologic study was made by Dr. Selinger, bacteriologist at the Episcopal Eye, Ear and Throat Hospital, Washington, D. C.

In order to exhibit the results, I present all the bacteriologic findings, although they are not identical in numbers as to each group.

It will be noted that the findings in each group correspond almost exactly as to the prevailing micro-organism, the staphylococcus being the most frequently present in each group. Contamination occurs in several cases. Cultures made in the hospitals showed less contamination than those made in my office. The contamination referred to is that of the hay bacillus.

BILATERAL PARALYSIS OF THE ADDUCTORS OF THE VOCAL CORDS OF ORGANIC ORIGIN

REPORT OF A CASE OF LANDRY'S PARALYSIS *

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The synonyms for bilateral paralysis of the adductors of the vocal cords are functional aphonia and hysterical aphonia, designating its functional origin. The present case is of interest as the bilateral paralysis of the adductors appears to be of definite organic origin. The case is unusual in another respect, in that it belongs to the class of cases described by Landry, and yet complete recovery followed.

REPORT OF CASE

M. B., a white woman, aged 29, was admitted to the obstetric service of the New Haven Hospital, complaining of vomiting and loss of weight. Seven weeks previously she had begun to vomit. Her family physician told her that she was three months pregnant, even though her periods had been regular. The last period had occurred one month before admission. Twelve days before admission the patient attempted an abortion, and bled for a week. Following this, the vomiting became more severe, until finally the patient was unable to retain any food.

Examination showed a markedly dehydrated patient with much recent loss of weight, but otherwise apparently normal. While a neurologic examination was not made, neurologic changes were not obvious. The patient also appeared mentally clear. She did not have elevation of temperature and her pulse ranged between 100 and 120. Even though the pelvic examination did not suggest pregnancy, the impression was that she had the neurotic vomiting of pregnancy. Under conservative treatment the vomiting slowly ceased, but the patient gradually became so hoarse that she could only whisper. She underwent a great mental change, and thirteen days after admission was transferred to the medical service, under the impression that she had a major psychosis. The temperature remained normal, but the patient developed a persistent tachycardia with a pulse rate of around 140. Examination by several physicians during the next three days brought out the following points:

The patient showed much mental confusion. The eyes had a wide open appearance, which did not appear to be exophthalmos, and a pronounced oscillation (not nystagmus) of both eyeballs was noticed. There was no definite ocular paralysis but there appeared to be a limitation of motion so that all of the extra-ocular movements were incomplete. The pupils were equal and regular, and contracted normally to light. The eyegrounds appeared negative. The voice was husky, and the swallowing of water caused difficulty and coughing, apparently from aspiration. Paralysis of the facial muscles was absent, and paralysis of the tongue or of the pharynx could not be noted. The lungs were normal, as was also the heart, except for a marked tachycardia, with pulse rate of 140. Subsequent electrocardiograms showed this to be a sino-aurcular tachycardia. Weakness of the upper or lower extremities could not be noted. While the abdominal reflexes were not obtained, all of the deep

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reflexes were readily obtained. Laboratory examination showed that the blood and urine were normal. The Wassermann examination of the blood and spinal fluid were both negative. The spinal fluid examination showed three cells.

The whole picture was very confusing. Superficially, it suggested hysteria, but the persistent tachycardia, with hoarseness and difficulty in swallowing, indicated a paralysis of some sort.

Twenty days after the patient's admission to the hospital, I had an opportunity to examine the larynx. The spoken voice was very weak, so that she could only whisper; she had great difficulty in coughing. There was no paralysis or anesthesia of the pharynx. The larynx was easily seen with the mirror (figure 1). The left arytenoid moved slightly on phonation, but the left cord remained in the position of abduction. The right cord adducted slightly on phonation but did not approach the midline. A large amount of mucus could be seen in the trachea, which it was almost impossible for the patient to expel. The patient's pulse at this time was 160. The oscillations of the eyes with limitation of extra-ocular movements still persisted.

The next day the patient appeared much worse. Her breathing had become more difficult; respirations were rapid and purely thoracic, and there was a sinking in of the abdomen with respirations, suggesting paralysis of the diaphragm.

Fig. 1.—Attempted phonation. The left cord in extreme abduction. The right cord adducts as far as the cadaveric position.

The patient was unable to cough. The lungs remained essentially normal. The heart showed no changes except the tachycardia as before noted. Both legs showed slight muscle weakness for flexion and extension. Deep reflexes were still active and the plantar reflexes were definitely the same. There was no loss of sphincter control. During the whole course of observation definite sensory changes had been absent. The larynx at this time showed a complete paralysis of the left cord which was in the cadaveric position. The left arytenoid remained fixed, while the right cord remained the same as the previous day. The patient was put on a course of strychnine and likewise was treated with digitalin. This was essentially the only treatment. She showed no change in her general condition for several days.

Twenty-five days after admission, the patient appeared somewhat better and her breathing seemed easier. She gradually developed less trouble in swallowing, but she was still unable to cough. Oscillation of the eyes had entirely disappeared. The vocal cords remained the same, so that she could barely whisper. For the next few days, she improved in every respect.

Thirty-five days after admission the patient phonated for the first time since her trouble began. Her general condition was greatly improved. The stuporous condition had entirely disappeared and she was able to sit up. Four days later, a change was noted in the larynx (figure 2). On phonation it was seen that the left arytenoid definitely moved, though the left cord remained in the cadaveric position while the right cord moved to the midline.

Forty-one days after her admission the patient was able to talk in a low whisper by an effort. The left cord was still in the cadaveric position, though it was tense and the arytenoid moved normally. The right cord approximated the left in the anterior third although not in its posterior two-thirds. The return of the voice coincided with the complete return of adduction of the right cord and partial adduction of the left. Accompanying this change in the larynx there was a definite decrease in the tachycardia, although the pulse rate still persisted around 110 to 120.

Two and one-half months after admission the patient was still in the hospital. She had gradually regained some of her strength and was able to get about,

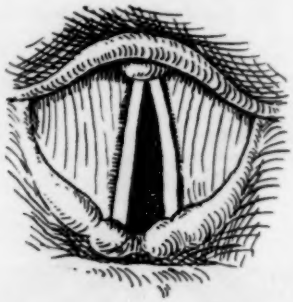

Fig. 2.—During phonation. The left cord adducts as far as the cadaveric position, while complete adduction has returned to the right cord.

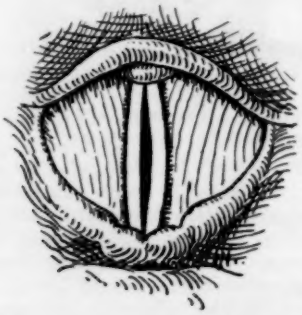

Fig. 3.—During phonation. The voice is still weak. Complete adduction has returned to both cords, but the left cord is relaxed and does not approximate the right one accurately.

although her legs were still weak. She could talk without effort, and, though her voice was weak, there was no hoarseness. Examination of the larynx showed the right cord normal (figure 3). The left cord appeared slightly thinner than the right. On phonation, complete adduction seemed to have returned to the left cord, but it did not approximate the right cord accurately. It appeared as if the tensor of the left cord was still paralyzed. Abduction was normal. The tachycardia had completely subsided, and the pulse rate was now around 90.

The patient was seen two months after her discharge from the hospital, that is, four and one-half months after her admission. She still had some weakness in her legs, which was noticeable only on going upstairs. She talked normally and her voice was loud and without tremor. On examination both vocal cords moved

normally on phonation and deep respiration. Her only complaint was a swelling of the neck; the thyroid gland had become noticeably enlarged. The patient was without toxic symptoms, however, and her pulse was 96.

One year after her admission to hospital, the patient reported that she was doing nicely. Her voice was normal, the weakness of the knees had entirely disappeared and the swelling of the thyroid had greatly subsided. Other symptoms had not developed. Examination of the patient was not made at this time.

COMMENT

From the point of view of diagnosis, this case was exceedingly interesting and problematic. At first, the picture was that of hysteria. An attempted abortion in a person of Catholic faith was sufficient cause for a hysterical reaction. The mental changes accompanied by the loss of the voice, which were the first symptoms, suggested hysteria, more especially since it was seen that the larynx presented the picture usually seen in hysterical aphonia. The one fact against hysteria was the marked tachycardia without fever. With the development of subsequent paralysis, a definite organic disease was evident. A toxic neuritis was ruled out, not only by the lesions present but also by a careful review of the history.

While not presenting the typical picture of Landry's paralysis, there are features which put it in this class and separate it from acute polyneuritis. The general symptoms were very slight, and the onset of the paralysis was gradual. There are cases of Landry's paralysis on record in which paralysis of the motor cranial nerves was the first symptom. In the present case there was paresis of the third, fourth and sixth cranial nerves and a definite paralysis of the vagus nerve. Later, the phrenic nerves were involved and apparently there was a paresis of the peripheral nerves to the arms and legs. Tenderness on pressure over the muscles, and sensory changes were not observed. The sphincters were not involved and the deep reflexes remained normal. Changes in the spinal fluid were not noted. While not a true ascending paralysis, these features seem sufficient to make the diagnosis of Landry's paralysis of the type beginning with bulbar symptoms. It is realized that Landry's paralysis is more of a symptom complex, and that it may be poliomyelitis or encephalitis.

Paralysis of the diaphragm in this case would be hard to prove. Inability to close the glottis in itself makes it difficult to cough. Such was the case during most of the course of the disease. Yet for a few days there was a decided change. Respirations were very rapid and were purely thoracic, and there was a sinking in of the abdomen with each respiration. It is interesting in this connection, for the production of massive collapse of the lungs is based on a paralysis of the diaphragm.

Of special interest were the changes in the larynx. A bilateral paralysis of the adductors of the vocal cords developed and subsequently

cleared up under observation. The paralysis was unequal and was accompanied by a tachycardia without fever, indicating a vagus paralysis.

History of any sort of respiratory infection preceding the onset was absent, and local changes were not noted in the cords or larynx other than the paralysis, which would be against a rheumatic condition. Gerhardt, Semon and Mackenzie have all seen somewhat similar cases, and each of these observers felt that when the paralyses were asymmetrical and associated with tachycardia the condition should be considered of organic origin.

In the present case, all the signs and symptoms of a typical case of hysterical or functional aphonia were given. Yet, occurring as it did, as part of a paralysis involving parts other than the larynx, its organic origin can hardly be doubted. Its resemblance to other cases which have previously been called functional aphonia suggests that possibly many other of the functional cases have a definite organic basis, and that functional aphonia should not be considered synonymous with bilateral adductor paralysis.

THE OCCURRENCE OF OTOSCLEROSIS IN THE ETIOLOGY OF PROGRESSIVE DEAFNESS*

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The serious menace of chronic progressive deafness and the frequency with which otosclerosis, spongifying of the labyrinth capsule, appears to be the underlying pathologic cause has impressed us more and more as the study of these patients, extending over a period of years, has served to verify the correctness of an earlier, more or less tentative diagnosis. In order to gain some definite idea of the actual proportion of such cases, we undertook an examination of the records of 1,000 consecutive patients who had consulted us in 1924. In this list we found 362 who had come because of ear symptoms, and of this number there were 113 for whom we had felt that otosclerosis was the only logical diagnosis; approximately one out of every three who consulted us because of ear symptoms was found to be suffering from this condition.

In considering these somewhat surprising figures, it should be borne in mind that in a practice that consists largely of patients referred by other physicians, it is to be expected that the number of those suffering from incurable conditions would be out of proportion to the number with temporary troubles, since the latter recover spontaneously or under the direction of the physician in charge.

ACCURACY OF DIAGNOSIS

A more important consideration in connection with such statistics is the question of the accuracy of diagnosis. This question is all the more pertinent because we have as yet no method for diagnosing otosclerosis from functional tests alone. When the spongifying of the labyrinth capsule develops in the region of the oval window, the first evidences that appear of a defect in hearing are those resulting from rigidity of the stapes. These symptoms are identical with those resulting from other pathologic processes producing rigidity in the sound conducting mechanism. On the other hand, spongifying of the labyrinth capsule may develop at a distance from the oval window when the symptoms produced are those of nerve deafness. Nerve deafness develops sooner or later in all cases of otosclerosis, but in the primary fixation of the stapes they are, in the beginning at least, relatively unimportant when compared with those produced by the stapes rigidity. The diagnosis of otosclerosis has, therefore, to concern itself, on the one hand, with the differentiation of primary fixation of the stapes due to changes in the osseous labyrinth from other conditions producing fixa-

* Read before American Otological Society at Montreal, June 4, 1926.

tion of the conducting mechanism, and, on the other hand, with the diagnosis of primary nerve deafness associated with spongifying of the bony capsule from other conditions producing nerve degeneration.

Of these two clinical types of otosclerosis, one causing primary fixation of the stapes and the other producing primary nerve degeneration, the former offers less difficulty in diagnosis, and from the post-mortem examinations that have been made appears to be the much more frequent type. "When the functional tests give the Bezold triad (elevation of the lower tone limit, prolonged bone conduction and a negative Rinne reaction) reactions characteristic of an obstruction in sound conduction, and when in addition, the examination excludes the presence of a tubo-tympanic process, that is, when the tube is found patulent and the drum membrane normal, the diagnosis of primary fixation of the stapes, the result of spongifying of the labyrinth capsule, seems assured." It is significant that not a single case has been reported in which the necropsy has failed to verify otosclerosis with fixation of the stapes when the foregoing clinical observations have previously been noted.

As a matter of fact, even before the pathology of this condition had been uncovered and when cases with functional reactions typical of obstruction in sound conduction were all diagnosed as middle ear processes, it had already been recognized that in those patients in whom the examination disclosed a patulent tube and a normal drum membrane the prognosis was especially bad, and that these were the cases particularly in which local treatment was of no value, and in which the deafness became progressively worse. It was recognized, too, at that time that the symptom of paracousis Willisi indicated an unfavorable prognosis, and that it was present usually in this particular type of obstructive deafness. Such a close observer as Politzer had pointed out that the pathologic condition in these cases must be restricted to the niche of the oval window, and they were often designated, therefore, as the circumscribed type of middle ear catarrh with the pathologic condition restricted to the niche of the oval window, as distinguished from the diffuse middle ear catarrh with alterations involving the tube and drum membrane as well. At this time the term otosclerosis began to be used to distinguish those patients in whom the drum membrane remained normal and quite transparent from the hyperplastic form of middle ear catarrh, which always produces palpable evidences of thickening and other alterations in the membrana tympani.

In the second type of otosclerosis, that in which the spongifying process develops at a distance from the oval window and in which the stapes remains movable, the functional conditions may be typically those of primary nerve deafness. This type is less frequently recognized and apparently occurs less frequently than that producing primary fixation of the stapes. That the spongifying of the labyrinth capsule

could be the responsible etiologic factor when the functional conditions were those of primary nerve deafness has been verified by necropsy. More extensive necropsy study of cases of primary nerve deafness, especially those in which the deafness begins in early adult life, may show that otosclerosis is more frequently the cause than we now suspect.

For obvious reasons it is palpably more difficult to make a differential diagnosis of otosclerosis when the clinical conditions are those producing nerve deafness than it is when the conditions are the result of stapes fixation. In the latter we have, on the one hand, all the tangible evidences of tubotympanic alterations to help in a differential diagnosis, whereas in the cases of primary nerve deafness we are left with only such evidences as can be gathered from the history of an insidious onset beginning in early adult life, the occurrence of other cases of genuine otosclerosis in the immediate family, and the absence of any other probable cause for the labyrinth involvement to assist in making the diagnosis. Occasionally the diagnosis of the spongifying process is made more definite by the presence of a pinkish glow from the promontory.[1]

TUBOTYMPANIC PROCESSES

We have considered so far only those cases of otosclerosis in which this process appeared as a primary ear lesion. That the spongifying of the osseous labyrinth may develop in patients with palpable evidence of tubotympanic alterations has already been verified by necropsy. The diagnosis of bony fixation of the stapes resulting from spongifying of the osseous labyrinth is, of course, not so readily made when alterations exist in the tube and drum membrane, indicating the presence of a middle ear process which may also be capable of producing the symptoms of obstructive deafness. An experienced otologist, accustomed to making a

1. In the Annals of Rhinology, Otology and Laryngology for March, 1903, one of us reported a case of this type in a young man, aged 27, whose father was found to have an advanced otosclerosis in which fixation of the stapes was combined with profound nerve degeneration and who some years later became totally deaf. This young man stated that he began suffering from tinnitus aurium between the ages of 3 and 4 and the insidious onset of deafness a few years later. The course of the trouble was followed for some five years, during which time the defect gradually increased, always retaining the clinical symptoms of nerve deafness—Rinne reaction positive, lower tone limit normal, defect in the upper tone range and shortened bone conduction. During all this time there was a marked pinkish glow from the promontory in both ears. Twenty years later he again presented himself, at which time he was using an electrical hearing device. This was surprising, since these devices are not of any particular assistance in cases of primary nerve deafness. An examination disclosed the fact that he had developed the typical reactions of stapes fixation—a negative Rinne reaction, marked elevation of the lower tone limit in which the a Bezold fork was not heard by air conduction, and a prolonged bone conduction. At the same time the defect previously noted in the upper tone range had increased very much so that the Edelmann-Galton whistle was not heard in either ear. The pinkish glow from the promontory had disappeared from both drum membranes.

careful study of cases of obstructive deafness caused by adhesive middle ear processes and those resulting from bony fixation of the stapes, will often be able to determine which of the two processes is responsible for the clinical symptoms even when they are associated in the same person.

Clinical experience has shown that tubotympanic processes, which occur with such frequency in early childhood producing permanent alterations in the membrana tympani, very often leave no permanent disturbance in hearing. The evidences of an exhausted tubotympanic catarrh or the existence of residue from an exhausted suppurative otitis media can hardly be expected to prevent the later development of a latent hereditary tendency for the spongifying process. The infrequency with which we are able to diagnose the otosclerotic process in the presence of these middle ear changes should not be interpreted as evidence that the alterations produced by a previous tubotympanic process in any way hinder the development of a spongifying process any more than that the existence of these two processes indicates that the spongifying of the capsule has been the result of the previous middle ear disease.

Considerations such as these render it unlikely that the skiagraphic study of the mastoid will aid materially in arriving at the diagnosis of otosclerosis. Evidence seems to indicate that arrested pneumatization of the mastoid is the result of early pathologic changes in the middle ear. That this arrested pneumatization renders the prognosis more serious when a subsequent middle ear infection develops seems probable, but that this arrested pneumatization protects the ear in any way from the subsequent development of a latent hereditary tendency for spongifying of the labyrinth capsule is quite another matter. Our studies of skiagraphy of the mastoid have shown that in cases of otosclerosis in which this process develops as a primary disease, that is, in which the membrana tympani remains quite normal, the pneumatization of the mastoid is usually complete. On the other hand, we have found arrested pneumatization in patients in whom from the manner of onset, the presence of other cases of otosclerosis in the immediate family, and the progress of the ear trouble, we were quite confident that otosclerosis was the cause. We are inclined to attach the same importance to the presence of arrested pneumatization, as shown in the skiagraph, as we do to the alterations in the drum membrane, both being indicative of some previous middle ear disease but neither having any relation to the subsequent development of otosclerosis.

One question that arises in the mind of anyone who has studied a large number of cases of otosclerosis is the significance of the unusual transparency of the membrana tympani in so many of the patients with a primary spongifying process. Is there anything suggestive of a pathologic condition in this unusual transparency of the membrana tympani? Our feeling is that this transparency is not indicative

of pathologic alterations. Its occurrence in the presence of the functional reactions of obstruction deafness gave rise to the term otosclerosis to distinguish these cases from the hyperplastic middle ear processes because it was believed that some middle ear pathologic change was responsible for all obstructive deafness. That spongifying of the labyrinthine capsule should be found so much more frequently in patients in whom the drum membrane remains typically normal is, of course, to be expected unless we assume, as some have done, that the development of otosclerosis is often the result of a middle ear disease. If we assume that the spongifying of the labyrinth capsule is the result of some general, as yet obscure systemic alteration capable of causing these local osseous changes, especially when there is an inherited tendency, we should of course expect that the vast majority of patients suffering from otosclerosis would show no evidence of any previous middle ear trouble, for the simple reason that there are so many more people who escape entirely any inflammatory middle ear disease capable of producing alterations in the drum membrane than there are those in whom such middle ear disease has occurred.

FUNCTIONAL REACTIONS

With these preliminary considerations of the problems concerned in making a diagnosis of otosclerosis, we shall present some of the facts that came out in our study of the 113 patients in whom the diagnosis of otosclerosis was made.

In ninety-two of these patients the drum membrane was typically normal. In a small number of these cases, in which the process was still restricted to one side, the drum membrane in the normal hearing ear showed such slight alterations as persist so frequently after an exhausted tubotympanic process. In fifteen of the ninety-two cases in which the drum membrane remained free from such alterations, a distinct pinkish glow was present from the promontory that we recognize as pathognomonic of otosclerosis since this particular alteration is never found in any other condition. It is observed usually in the earlier stages of this process and persists for a period of years but eventually disappears entirely. It has sometimes been looked on as an evidence of a particularly active stage of the spongifying process. We suspect, however, that its disappearance may more likely be due to the fact that the spongifying over the immediate region of the promontory has been completed while the active process has extended to other parts of the osseous capsule; its presence, therefore, is not an evidence of a more active stage but rather of an active involvement of a particular region, the promontory. This permits of its being detected by otoscopic examination.

In twenty-one cases the diagnosis of stapes fixation was made in the presence of palpable alterations of the drum membrane indicating an

exhausted tubotympanic process. As already pointed out, such alterations in the drum membrane usually indicate a condition which developed in early childhood and which rarely leaves any permanent defect in hearing. For obvious reasons the diagnosis of otosclerosis is made in such cases only when the clinical evidences of a profound fixation of the stapes are out of all proportion to the fixation which the slight alterations on the drum membrane would lead one to anticipate; when from the insidious manner of onset, the absence of any active tubal process, and the occurrence of other typical cases of otosclerosis in the immediate family make the diagnosis of spongifying of the bony capsule the only logical conclusion. The fact that such patients have already had a tubotympanic process leaving palpable changes in the drum membrane is, of course, no reason for believing that this condition will prevent the later development of the spongifying any more than would an arrested pneumatization of the mastoid.

In 105 of the 113 cases of otosclerosis the functional reactions were those resulting from stapes fixation, that is, the reactions of an obstruction in sound conduction. In all these cases there was also a more or less extensive lowering of the upper tone limit. In the advanced cases this evidence of nerve degeneration became quite extensive and occasionally extensive internal ear involvement was found associated with only a moderate elevation of the lower tone limit, indicating only a partial rigidity of the stapes.

In contrast to these cases giving the reactions characteristic of stapes fixation and nerve degeneration is the second type of otosclerosis, in which the stapes is not involved and in which the functional tests are those typical of a primary nerve deafness. As we have already pointed out, the diagnosis of such cases is much more difficult than that of primary stapes fixation. The diagnosis of otosclerosis was made only when from the manner of onset, the period of life in which the process developed, the absence of any other likely cause for nerve deafness, and especially the occurrence of other cases of otosclerosis with stapes fixation in other members of the immediate family, the diagnosis of spongifying seemed the only logical conclusion. Occasionally in these cases the pinkish glow from the promontory gave substantial aid in reaching this conclusion. Altogether our series included only eight cases with the functional reactions of primary nerve deafness. We suspect that this figure hardly represents the true proportion of these cases, and that more extensive necropsy study of patients who have suffered from primary nerve deafness may show spongifying of the osseous labyrinth as a more frequent cause for this type of deafness than we now suspect.

It has been pointed out that vertigo often occurs, especially in the later stages of otosclerosis. We were rather surprised that this symptom

was recorded in only six of the 113 cases. We had suspected a much more frequent occurrence of active symptoms of involvement of the vestibular mechanism. We think it possible that the symptom was not recorded in some cases in which a more careful questioning might have brought out the fact that this had existed in the past. Turning tests for detection of alterations in the vestibular mechanism were made in a good many patients, but in only very few was there any deviation from normal. This subject is one which should receive further study.

The symptom of paracousis Willisi is such an important one in patients suffering from otosclerosis that we examined our records to determine its frequency. Paracousis Willisi, the ability to hear better in a noisy place, is not detected in the earlier stages of otosclerosis. It is only when the defect in hearing becomes marked that this symptom comes into prominence. For obvious reasons it is not present so long as the process is restricted to one ear. Ability to hear well over the telephone appears to be usually present when paracousis Willisi exists.

The two phenomena are not dependent on the same cause, as is evidenced by the fact that they may occur independently of each other. The symptom of paracousis can be explained as the result of an elevation of the voice, usually quite unconsciously, in the presence of external sounds. In stapes fixation the voice is more easily heard in the presence of external sounds because the fixaion succeeds in filtering out most of these external sounds, leaving the perception for the voice unhampered; a person with normal hearing experiences increasing difficulty in hearing the voice as the external noise increases. The ability to hear well over the telephone is dependent on the phenomenon of bone conduction. Since bone conduction is usually heightened when stapes fixation exists, the phenomenon of paracousis and the ability to hear well on the telephone usually coexist. In primary nerve deafness paracousis is absent because there is no mechanism for filtering out the external sounds. As a matter of fact, most patients with primary nerve deafness experience difficulty in using the telephone because in most of these cases a diminished bone conduction exists. Occasionally we observe a person with primary nerve deafness, that is, one in whom the defect is chiefly in the higher tone range and in whom the perception for the lower part of the scale is unimpaired, in whom for some as yet unknown reason the bone conduction is unimpaired or actually increased. These patients experience little difficulty in hearing over the telephone and there is a complete absence of the symptom of paracousis.

We found that paracousis Willisi was distinctly recognized by sixty-four patients. In twenty-nine our records show that it was not recognized and in twenty no notation was made regarding this symptom. The latter number should be added to those in whom the symptom was not present since we often made no notation when there was an absence of

a symptom. There were then forty-nine patients in whom the symptom of paracousis Willisi had not been complained of as opposed to sixty-four in whom it was present. In view of the fact that paracousis is never a symptom in unilateral involvement and is only present when there is a more or less profound defect in hearing, the fact that it was present in sixty-four out of 113 patients was rather striking. Although it is a symptom related to obstruction in sound conduction, it is not so often observed when this obstruction is the result of a plastic adhesive middle ear process, such as that left from an exhausted chronic suppurative otitis media. It is almost pathognomonic of the spongifying process with fixation of the stapes.

SUMMARY

1. Of a total of 113 cases diagnosed as otosclerosis, in ninety-two the drum membrane was quite normal. In fifteen there was a distinct pinkish glow from the promontory. Twenty-one patients showed some alterations in the membrana tympani. In 105 the functional tests demonstrated fixation deafness. In eight patients the functional reactions were those of primary nerve deafness. Paracousis Willisi was recorded in sixty-four patients. Complaints of vertigo were obtained in six patients.

2. The clinical recognition of this pathologic entity is limited for the most part to those cases in which the process is a primary lesion, that is, to those in which it is not preceded by any tangible evidences of a previous tubotympanic inflammation.

3. The type of otosclerosis usually diagnosable clinically is the type which causes fixation of the stapes and in which no evidences of previous tubotympanic trouble exists.

4. The recognition of otosclerosis as the cause for stapes fixation can often be made even when tangible alterations exist in the drum membrane indicative of an old tubotympanic process, since the two usually do not run the same clinical course.

5. Otosclerosis develops also with the symptoms typical of nerve deafness, that is, in cases in which stapes fixation is absent. The diagnosis of this type is obviously more difficult.

6. It seems not unlikely that because of this difficulty in making a diagnosis, further necropsy observations may prove that spongifying of the labyrinth capsule in the absence of stapes fixation may be responsible for the occurrence of primary nerve deafness more frequently than we now suspect.

7. An associated degeneration of the nerve, as evidenced by the defect at the upper end of the tone scale, develops more and more as otosclerosis producing stapes fixation progresses.

8. Otosclerosis without fixation of the stapes, beginning with the symptoms typical of nerve deafness, may as time goes on develop stapes fixation with the typical Bezold triad superimposed on the previously existing nerve deafness.

9. A pinkish glow from the promontory is pathognomonic of otosclerosis.

10. The symptom of paracousis Willisi is rarely found except in cases of otosclerosis with stapes fixation.

11. Skiagraphy of the mastoid does not appear to be of any particular assistance in fixing the diagnosis of otosclerosis. Normal pneumatization can be anticipated when all evidences of a previous tubotympanic process gathered from examination of the drum membrane are absent. Arrested pneumatization has the same significance as palpable alterations in the drum membrane. Both are indicative of an earlier inflammatory middle ear disease. The presence of arrested pneumatization, just as the existence of alterations in the drum membrane, can hardly be expected to interfere with the subsequent development of the spongifying of the labyrinth capsule.

CONCLUSIONS

Otosclerosis is the most frequent cause for those cases of progressive deafness which result in a more or less severe handicap. The pathologic changes in the ear that cause the impairment of hearing are always in the nature of fixed or degenerative changes which cannot be altered by any form of treatment, local or general. Patients suffering from otosclerosis are the frequent victims of ruthless exploitation at the hands of medical quacks. Such exploitation has increased greatly in recent years and the extent to which it has proceeded, promoted by widely distributed bulletins and often by misleading articles in the lay press, may be surmised from the variety of bizarre forms of local treatment with which these unfortunates are constantly being pursued by men who cultivate the faculty of salesmanship in place of professional skill and common honesty.

This exploitation takes the form of treatment by attenuated irradiation of the pituitary body, local applications and manipulations by way of the eustachian tube, and the so-called "finger surgery" in the nasopharynx—all equally useless. Elaborate and expensive electrical devices are constructed for the purpose of restoring, by the prolonged application of loud sounds, the function of a conducting mechanism out of commission because of bony ankylosis, and of a nervous mechanism that has undergone degenerative changes. Another "cure," widely advertised both through special bulletins and by articles in the daily papers, consists of an effort to commercialize the phenomenon of para-

cousis Willisi. A rather elaborate electrical apparatus is devised for producing vibrations below the threshold of hearing in order to develop the phenomenon of paracousis Willisi so common in otosclerotic patients, and the hope is held out that by the prolonged and repeated application of these vibrations a permanent improvement in the hearing will result.

The first method, that of prolonged bombardment of the ear with very loud sounds, ignores a long established fact, that this is one of the surest methods of producing degeneration in the cochlea. The second, the attempt to improve the hearing permanently by stimulating the symptom of paracousis Willisi, overlooks the fact that otosclerotic patients whose occupations subject them continually to surroundings that develop paracousis Willisi proceed, nevertheless, exactly as do all these patients, with the progressive loss of hearing.

It may be expecting too much of manufacturers to look to them to seek cooperation and approval from scientific otologists before elaborating expensive devices, the only result from which is the victimizing of people handicapped by incurable deafness. We should endeavor to develop among the general medical profession a better appreciation of the fundamental facts regarding otosclerosis in order to gain their cooperation in the obligation that is ours of protecting so far as may be possible the victims of this form of incurable deafness from the ruthless exploitation of medical quacks.

Instruments devised as aids for hearing do not come under the type we are here criticizing. The solution of the clinical problems of otosclerosis lies along the further perfection of instruments of this type, and the prevention of the development of spongifying by a better knowledge of the factors underlying its cause.

REACTIONS INDUCED THROUGH THE DUCTUS ENDOLYMPHATICUS AND THE AQUEDUCTUS COCHLEAE

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Gruber,[1] in 1890, spoke of the effect of increased intralabyrinthine pressure being transmitted through the saccus endolymphaticus to the cerebellum as a cause of vertigo.

In 1913, I [2] called attention to the saccus endolymphaticus as a safety valve for increasing intralabyrinthine pressure.

Portmann,[3] in 1921, experimenting with the ductus endolymphaticus in selaciens, which in them opens on the exterior, near and a little behind the eye, found that this fish after closing these openings by cauterization, acted, for a certain period of time, as though the static labyrinths had lost their functions.

At the meeting of the Eastern Section of this society, January 23, of this year, I explained labyrinthine tonus as the result of intracranial pressure waves transmitted directly to the vestibular nerve end organs through the ductus endolymphaticus.

The membranous labyrinth is a closed series of cavities which act as receptacles for the nerve terminations of the cochlear and vestibular nerves and the endolymph.

The saccus endolymphaticus is a relatively large, flattened cavity lying on the posterior surface of the petrous pyramid between two layers of thin dura and subject to pressure changes in the posterior cranial fossa.

Pressure changes here are transmitted through the ductus endolymphaticus to the immediate vicinity of the maculae acusticae and the cristae ampullares.

The perilymphatic series of cavities in the labyrinth communicate directly with the subarachnoid space mesial to the jugular bulb, in the posterior cranial fossa, through the aqueductus cochleae.

Changes in the intracranial pressure are therefore transmitted to the perilymphatic cavities through the aqueductus cochleae.

Intracranial pressure increases with the cardiac systole, the expiratory phase of respiration, the Traube-Hering vasomotor arterial waves, with pressure over the great vessels of the neck (the Queckenstedt

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1. Gruber, Joseph: Diseases of the Ear, New York, D. Appleton & Company, 1890, p. 528.

2. Hubby, Lester Mead: A Discussion of the Modus Operandi of Galvanic Nystagmus, Ann. Otol. Rhin. & Laryng., Sept. 1913.

3. Portmann, Georges: Recherches sur la physiologie du sac et du canal endolymphatiques. Valeur fonctionnelle de l'organe endolymphatique des Sélaciens, Compt. rend. Soc. de biol. **85**:1070, 1921.

phenomenon), with every marked change of body position, with sufficient striated muscular effort and with mental conditions of anger, excitement, etc. In other words, it occurs over ninety times a minute with the body and mind in repose, or once in at least every two-thirds of a second.

The cranial and spinal cavities are filled with soft structures and fluid. They are practically incompressible. Relief of pressure comes through the emptying of the veins, of the cerebrospinal fluid and the lymph in the lymph spaces.

The labyrinth is also filled with soft structures and fluid and is also practically incompressible except at the two labyrinthine windows, where the membrana secondaria and the foot plate of the stapes freely move on pressure within certain limits.

The cerebrospinal pressure waves pass by two paths into the cavities of the labyrinth.

The endolymphatic pathway starts in the saccus endolymphaticus, proceeds through the ductus endolymphaticus and the ductus utriculo saccularis to the saccule, utricle, semicircular canals and from the saccule to the ductus cochlearis through the ductus reunions.

The perilymphatic pathway starts in the subarachnoid space in the posterior cranial fossa, proceeds through the aqueductus cochleae to the scala tympani close to the elastic membrana secundaria, passes through the helicotrema to the scala vestibuli, the vestibule and the semicircular canals.

This wave is also transmitted through the membranous walls of the ductus cochlearis to meet the synchronous wave from the ductus reunions, and so neutralizes it.

Likewise the perilymphatic waves on reaching the vestibule are met and neutralized by the endolymphatic waves passing outward through the walls of the membranous labyrinth into the perilymphatic cavities of the vestibule and semicircular canals. The elasticity and yielding qualities of the membrana secundaria and the footplate of the stapes and annular ligament in the oval window, permit these infinitesimal waves. Of course these wave currents are not entirely in one direction. Increased pressure is followed by a subsidence, so that it is a to and fro current. But the increased pressure waves are more abrupt than the subsiding pressures; just as in the pulse, the systolic wave is abrupt and the subsidence is more gradual like lysis.

Therefore the vestibular nerve terminations must be mainly affected by the more sudden, increased pressure waves.

LABYRINTHINE TONUS

The striated muscles of the body are constantly in a certain minimal contracting condition.

Severing the posterior spinal nerve roots causes a marked lowering of muscle tonus, showing that it is dependent on nerve control, that is, neurogenic rather than myogenic. In destruction of both labyrinths this tonus is lowered temporarily at least. The same is true of the functional destruction of the cerebellum. In the latter instance, however, the destruction of one side of the cerebellum produces lowering of the tonus on that side only. This is probably because the tonus efferent impulses pass through the cerebellum.

The changing intracranial pressure waves passing through the ductus endolymphaticus to the immediate neighborhood of the maculae acusticae and the cristae ampullares could readily initiate afferent impulses to the vestibular nerve centers, thus producing labyrinthine tonus.

Since these waves through the ductus endolymphaticus occur in both ears at the same time in the normal person, nothing more results than labyrinthine tonus effects on the muscles.

Each labyrinth exerts this tonic effect on all the voluntary muscles of the body.

VERTIGO

Vertigo will result from a sufficient and sufficiently sudden change in the relative amounts of nerve impulses from the vestibular nerve endings in the two labyrinths or in one labyrinth if the other labyrinth does not function.

As shown above, there is a constant stream of waves entering the membranous labyrinth through the ductus endolymphaticus. In increased intracranial pressure, these waves in the membranous labyrinth are greater. The stronger these waves, the greater the difference between those in the two labyrinths if the ductus endolymphaticus is suddenly sufficiently occluded on one side.

In bending the head forward and downward and to one side, as in putting on one's shoes, the cerebellum, being of higher specific gravity than the cerebrospinal fluid, comes in closer contact with the saccus endolymphaticus of that side and more or less blocks off momentarily its ductus endolymphaticus. This produces a relatively increased flow of waves in the duct of the other side. Add to this the increased intracranial pressure caused by flexing the neck strongly against the collar or neck band, and you have an ideal condition to produce vertigo or nystagmus in abnormal conditions of the vestibular nerve endings.

In toxemias of various kinds, whether bacterial or otherwise, there may be a hyperesthesia of the vestibular nerve shown in a hyperphysiologic nystagmus. In these conditions any slight sudden variation of the intraductus endolymphaticus waves of one side might cause vertigo or nystagmus. This would be especially true if the hyperesthesia were greater on one side than on the other.

Increased intracranial pressure, which also means increased intralabyrinthine pressure, may also be a cause of hyperesthesia of the vestibular nerve endings in the labyrinth.

There are great variations in different persons in the size of the recessus Cotugni which lodges the saccus endolymphaticus (Gruber). It may well be that for this reason the duct may be more easily occluded by pressure of the cerebellum in one person than in another, explaining the tendency to vertigo in these particular persons.

In labyrinthine disease there is a marked change in the afferent nerve waves from one labyrinth, with the result that nystagmus and vertigo occur.

The stage of stimulation, shown by nystagmus toward the affected labyrinth, is probably due to toxic or pressure irritation of the vestibular nerve endings on that side. The stage of paralysis, shown by nystagmus toward the other labyrinth, may be due either to loss of function of the vestibular nerve terminations of that side or to suppression of the normal intramembranous labyrinthine waves, or both.

Tympanic abnormalities that change from time to time may cause vertigo. This is brought about by changes in the intralabyrinthine blood circulation due to irritation of the sympathetic nerves in the tympanum and membrana tympani. In the same way a strong irritating liquid in the middle ear may cause nystagmus and vertigo (John R. Page). Sudden changes in the arterial supply of one labyrinth alter the endolymph currents in the membranous labyrinth, sufficient to upset the equilibrium between the two afferent nerve impulses from the labyrinths with the result that vertigo may ensue.

Should these changes happen to a neuresthenic person or to a person with hyperesthesia of the vestibular nerve endings, a smaller than normal threshold stimulation would bring about vertigo.

In many cases of tubotympanic otitides, toxins are present in the blood which might easily cause hyperesthesia of the vestibular nerves and so make the onset of vertigo probable.

BONY ANKYLOSIS OF OVAL AND ROUND WINDOWS

A cavity enclosed in unyielding walls, containing fluid and a nerve organ such as that of Corti, cannot vibrate sufficiently to ordinary sounds to produce auditory nerve impulses.

Sound waves are transmitted to Corti's organ through the labyrinthine windows and through the bony capsule.

The elasticity of the coverings of the two labyrinthine windows permits vibrations of greater amplitude than would be possible without such yielding structures.

If the round and oval windows have been filled in with solid bone, as in some cases of otosclerosis, it would seem that there should be

a total loss of hearing, which is not the case. This may be explained by the fact that there are two tubes, the aqueductus cochleae and the ductus endolymphaticus, and blood vessels entering this cavity, which permit some vibrations of the endolymph and thus a slight stimulation of Corti's organ.

The elasticity of the foregoing takes the place of the greater normal elasticity of the coverings of the round and oval windows.

SAFETY VALVE ACTION

The saccus endolymphaticus is ideally placed to act as a safety valve for increasing intralabyrinthine pressure. It is outside of the labyrinth and lying on a bed of loose areolar-like tissue. In the dog, according to Portmann,[4] it is nearly twice as large as the saccule, and the walls are creased in certain spots making it capable of enlargement.

Increasing intralabyrinthine pressure causes the squeezing out of the lymph and blood in this tissue as well as acting to decrease the cerebrospinal fluid and the blood in the veins and venous sinuses of the cranial cavity.

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4. Portmann, Georges: Recherches sur le sac et le canal endolymphatiques du chien, Compt. rend. Soc. de biol. **83**:45, 1920.

RESULTS OF TONSIL OPERATIONS

A QUESTIONNAIRE REPORT *

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Recently, having occasion to go through the literature, I was surprised to find so little, and that very scattered, on the subject of after results of tonsil operations.

I sent out 200 questionnaires to physicians in every state in the Union, and received 146 answers from every state excepting three, namely, Arizona, Connecticut and Idaho.

These answers, therefore, give the general opinion of the physicians in this country. The percentage given is, of course, from memory.

Fourteen questions were asked and were answered as follows:

Question 1.—Have you ever seen bad results when the uvula has been removed? If so, how much of it was removed and what were the symptoms?

Seventy answered just "no." One answered just "yes." Six said temporary symptoms only when part or all was removed.

Sixty-nine answered "no," when all or part only was removed, and "yes," when part of the soft palate was removed with the uvula.

The symptoms complained of were change of voice, some difficulty in swallowing and fluid passing through the nose, together with a dry pharynx. I have seen no bad results when only the uvula has been removed. I have seen two patients in whom the tension from contractions was so great that the uvula was drawn into the soft palate.

Question 2.—Have you seen cases in which the palate was paralyzed so that fluid came through the nose? If so, how long after the operation did it start and how long did it last?

Fifty-seven answered just "no." Eighty-one answered "yes." The paralysis comes on immediately, or three to four days later, and lasts from seven to ten days. In a few, the condition lasted from one to six weeks. Many claimed that the condition was not due to paralysis but to swelling of the soft palate, preventing closure.

One physician stated that, in removing large adenoids, the palate needs to readjust itself, due to the large space left.

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* Read before the Section on Laryngology, Otology and Rhinology at the Seventy-Seventh Annual Session of the American Medical Association, Dallas, Texas, April, 1926.

One man, in 5,000 operations, saw eighteen subjects in whom symptoms began on the third day and lasted from four to five days. In one, the symptoms began on the seventh day and lasted thirty days.

Three physicians had patients in whom the condition lasted three months; in one it lasted six months, and three still have trouble after three to five months.

In one man, the symptoms came on eight days after operation, but it was discovered that he recently had diphtheria. Diphtheria might be the cause of prolonging the condition.

Question 3.—Have you seen patients in whom the voice was changed after an operation? If so, how do you explain it?

Thirty-one answered just "no." Three answered that the change was only temporary.

Seventy-six answered that the voice was better if the pillars were not injured. This change was due to increased resonance by removing the obstruction caused by large tonsils—getting rid of pharyngitis and laryngitis caused by extension from tonsillitis. There was freer movement to the pillars.

Twenty-two commented on operations on singers. The speaking voice was not changed, but the singing voice had improved, giving better higher tones and a wider range; these tones were obtained easier and the voice was less tired.

In four cases the upper tones increased from one to two notes, but no change was noted in the lower tones.

I had a patient who lost one upper note but gained two lower notes.

One case reported was of a baritone with the anterior pillars injured on one side and considerable scarring, who lost the higher tones, but these were restored with voice exercise.

Fourteen commented on changes. Some patients claimed they were unable to sing after operation; others claimed their friends said their voices were changed, or, as one man explained it, the voice was never real.

In two cases reported, the patients were unable to talk without becoming hoarse. In five cases, the voice was impaired with the throats apparently perfect. In one case, there was almost complete aphonia (an actor), which the physician was unable to explain. One case followed a secondary operation (probably due to scar).

Question 4.—Have you seen infection in the fossae following operations? If so, how severe were they and what was your treatment?

Thirty-two answered just "no." Eighty-six answered "yes."

In most cases the infection is mild, and lasts from three to four days. A few quite severe conditions lasted from seven to ten days. One patient required two to three weeks treatment.

One physician believed that all patients are at least slightly infected. Another has never had an infection since swabbing the throat with iodine solution immediately after operation.

The common treatment was hot gargles, painting the throat with iodine solution, silver nitrate (2 per cent to pencil), mercurochrome-220 soluble and argyrol.

Nineteen patients have had abscesses needing opening following operations. Five had abscesses following the needle puncture in local anesthesia. In three, an abscess in the fossae above, in one, an abscess in the fossae below, and in two, an abscess in the pharynx were present. Eight patients had deep abscesses which were opened externally.

Nine patients had the following complications:

In four, Vincent's angina was present, which was fatal to one patient in two weeks; in one, ulceration was present, the patient had syphilis; erysipelas was present in one; two had diphtheria, and one patient had tuberculosis, which proved fatal.

One physician believed that all secondary hemorrhages were due to infection. I believe that all patients with a thick, heavy membrane or scab are infected. In all, improvement is rapid when treated with iodine.

Question 5.—Have you seen granulation tissue form in the fossae? If so, in what percentage of cases?

Fifteen answered just "no." One hundred and eighteen answered "yes," varied from rarely to nearly all cases, or 0.5 to 25 per cent; most said in from 5 to 10 per cent.

One man said 50 per cent in children, 10 per cent in adults; four said 90 per cent, two said 30 to 40 per cent and one said 60 per cent.

Two answered that this condition was very common in adults; one said it was noticed when the dissection was too deep. One said when portions of the capsule were left; another said it was due to muscle injury.

I believe granulation tissue forms in at least one-half of the adult patients and rarely forms in children. Generally, in those in whom the fossae were infected, the contractions are greater on healing.

Question 6.—Have you seen the lingual tonsils at the angle of the tongue turn into the fossae after the operation? If so, in what percentage of cases?

Twenty-five answered just "no." One hundred and fifteen answered "yes." A few said quite frequently; the other answers varied from 1 to 90 per cent.

One physician said it occurred in all patients in whom the plica is not removed. One said it was the infratonsillar lobe. Another said it was a piece of the faucial tonsil sometimes adherent to the tongue.

One said it was a plical hypertrophy, and two men said it occurred in nearly all adults unless care is used to remove it at operation.

I believe that, particularly in adults, there is in a great many, a small tonsil just below the tonsils proper or a large lingual tonsil coming off the angle of the tongue, which, if not removed at the time of operation, turns up into the fossae.

Question 7.—Do you believe in removing all this lingual tonsil at the time of the operation?

Eighty-five answered just "no." Forty-eight answered "yes." One physician always removes this tonsil in adults, never in children. Two men always remove this tonsil in children, never in adults. Eight men never remove tissue close to the tongue because of contractions. Two men never remove it at the time of operation unless it is large, but prefer to remove it later if necessary.

I believe that considerable care and judgment must be used in removing this tissue at the time of operation. Great contractions between the angle of the tongue and the pillars can result if too much tissue is removed in this locality.

In case of doubt, it is better to leave, and remove later if necessary, after the throat has completely healed. The amount of contraction is greatly lessened by this procedure.

Question 8.—Have you seen any severe contractions at the angle of the tongue following this procedure?

Sixty-three answered just "no." Six failed to answer. Seventy-seven answered "yes."

The answers varied from one to many, and from slight symptoms to severe, but there were no other comments. One patient had trouble for years afterward, and in another the symptoms lasted for a year.

I have seen some severe contractions, and at least two patients in whom the base of the tongue was drawn up so that the opening between the tongue and soft palate was very small.

Questions 6, 7 and 8, in regard to the lingual tonsil, were poor questions. Some confusion was shown by the term "lingual tonsils." Some spoke of a mass of lymphoid tissue just below the tonsil proper or just behind the plica, and others as a true lingual tonsil coming off the angle of the tongue.

I was thinking of the result, after an apparently good tonsillectomy, of finding, on healing, a mass in the lower fossae opposite the tongue, and if it was good surgery to clean out this area completely at the time of operation or to leave it and remove it later if necessary.

Question 9.—In what percentage of cases did the pillars retract more than you expected? Is this due to the predisposition of the patient, or, if not, what did you think was the cause?

The percentage, of course, was a guess and varied from 1 to 75 per cent; others said about 10 per cent and some answered from a few to a great many.

Twenty-three were unable to explain the cause. Twenty-five said it was due to faulty operations removing muscle fiber. Seventeen said this was due to injury to the posterior pillars; thirteen said to predisposition of the patient. Four said cutting too close to the superior constrictor bed caused this. Three said it occurred in patients in whom the tonsils were excessively large; three said it occurred in patients operated on too soon after quinsy; three said it occurred more often after the dissection method; one said it occurred more often after the Sluder method, due to removal of a small portion of the anterior pillars.

Two men had seen none, and three always expected contractions. One said it occurred in patients in whom effort to stop hemorrhages had been used. One said syphilis may account for some irregular results. The remainder gave indefinite answers.

In operations in which muscle tissue is not removed, I believe it is due, first, to predisposition of the patient; second, to low grade infection, and third to granulation tissue.

Question 10.—In patients in whom the pillars have been removed (either anterior or posterior), have you seen any bad postoperative symptoms? If so, what were they, and how long did they last?

Sixty-two said just "no," or did not answer the question. Forty-two said "no," when only the anterior pillars had been removed, and "yes" when the posterior pillars had been injured.

The symptoms varied. The one complained of most was a change in the voice. Some patients felt a pulling or drawing sensation on swallowing, chronic pharyngitis or dryness in the throat, reflex symptoms to the ears, and there were a few in whom food or fluid passed through the nose. Neurotic patients made the most complaints.

Thirty-three said "yes," and spoke of the length of time. Four said symptoms were permanent. Some said from one to several months, others, one to several years, but did not state if the recovery was complete. Six had seen patients with pillars all or partly removed, and the patients made no complaint.

One physician is suffering from atresia of the eustachian orifice, the result of contractions of scar tissue, but he does not state the site of injury.

I have seen many patients in whom the pillars have been all or partly removed; some have symptoms and many have apparently none at all. However, I believe that all have some symptoms, but that many adjust themselves to them.

Question 11.—In patients in whom the pillars or soft palate have been severely injured, followed by considerable scar tissue, of what symptoms have the patients complained?

This question had the same answers as question 10. Distinction was not made between removing just pillars and a condition in which the soft palate was involved.

Four spoke of adhesions of the soft palate to the pharynx, and one a case of complete atresia. I have seen one case of adhesion of the soft palate to the pharynx.

A few physicians spoke of a change in the taste. Patients called it a queer taste; its duration was short.

Question 12.—In what percentage of patients have you seen the adenoids return large enough to cause symptoms other than ear conditions?

Thirty-nine said none, or did not answer. Twenty-three said in very few cases. Fifty-one said from 0.5 to 10 per cent, more answered from 2 to 5 per cent. Two said 20 per cent; one said 50 per cent; six said never if properly removed; two said never if the tonsils were completely removed. Fifteen said from 1 to 25 per cent in children under the ages of 3 to 5 years.

One said the condition practically always recurs in children under 5. Two said it was due to postnasal discharge from sinusitis. Two questioned if the adenoid tissue ever was all removed.

I believe the nasopharyngeal wall is not smooth in all cases, and that this unevenness allows some adenoid tissue to remain which may enlarge; it is due probably to repeated colds or to sinus discharges and is more likely to recur in children under 5 years of age.

Question 13.—What is your usual after treatment for tonsillectomy in children and in adults?

The after treatment was quite universal. Most men had no after treatment for young children except to keep them in bed from one to three days. The bowels were kept open, and oil was used in the nose; two physicians had the children chew gum.

All adults used gargles of various kinds, powdered acetyl salicylic acid, acetphenetidin or orthoform was dissolved on the tongue for painful swallowing. It was surprising how universal was the use of acetyl salicylic acid, mostly as a powder on the tongue but also as a gargle.

In nearly every case beginning on the first to third day, the fossae were painted. The drugs used were argyrol, mercurochrome-220 soluble, silver nitrate, or iodine in various strengths.

Three physicians gave no after treatment, claiming the less manipulation the better.

Sixty-seven said no after treatment was given.

My after treatment is as follows:

Because it is difficult to handle young children, I give no treatment to the throat. The bowels are kept open, the children are allowed to eat anything they can swallow, and they are urged to chew gum to keep the jaws moving and to encourage swallowing. They are advised that the throat will be more sore and painful on the third or fourth day, as healing starts then.

Adults are given the same treatment, with the addition of a hot solution to hold in the back of the throat the first thing in the morning, and before eating to relax the tissues. If there is a great deal of pain on swallowing, powdered acetanalid or acetyl salicylic acid can be dissolved on the tongue.

On the third day the fossae are painted with tincture of iodine. If the scab or membrane is thick, it generally indicates a low grade infection, and the iodine is applied each day for four or five days. After this, 50 per cent of silver nitrate is used if granulations appear.

Question 14.—What is the average time of recovery?

This had a universal answer, children recovered, as a rule, a few days earlier than adults. Nearly all said a week to ten days; many said one week but most said two weeks. Some said a complete recovery took from two weeks to a month.

One man reported 150 cases, ranging in age from infants to 60 years, in which the earliest recovery was nine days; the longest fifteen days, the average being twelve days.

I see many patients who have been lead to believe that their throats will be well from three days to a week, and I think it is the duty of the throat specialists to dispel this idea.

COMMENT

In reviewing the answers to these questions, several interesting facts are brought out.

First, physicians, as a rule, are not very close observers of the after results of their work, or do not find out the reasons for their poor end-results.

Second, in answering questions they do not confine themselves to the subject.

Third, so far as results go, the tonsil operation, in many cases, is not satisfactory. True, the patient is rid of his repeated sore throat or focal infection, but in their place are often added new and different symptoms hard to relieve.

Fourth, even the most careful operator and the most perfect operation often leave after symptoms that are unexplainable.

Fifth, the predisposition of the patient as to scarring and his ability to readjust himself is an important factor.

Sixth, there is a great question in the minds of most men on the amount of tissue to remove at the base of the tongue at the time of operation.

Seventh, most men believe adenoids are likely to recur in children operated on before the fifth year.

Eighth, the after treatment and duration of time of healing is quite universal.

Ninth, that, on the whole, the good results obtained more than offset any occasional bad symptoms.

Tenth, that a physician should try not to express an opinion on a throat with an apparently bad result until he has made a very careful and exhaustive study of that particular case.

Eleventh, I wish to call attention to the anatomic variations in the pillars, on which subject I wrote a paper in 1924. One must not be mislead in what is really only an apparent deformity.

Twelfth, that a tonsil operation should be classed as a more major operation. If it were, physicians would be more careful as to their training before attempting it.

ABSTRACT OF DISCUSSION

DR. S. E. ROBERTS, Kansas City, Mo.: Dr. Pratt's paper should be regarded as a questionnaire on definite, probable and possible casualties. He certainly does not extol the virtues of a tonsillectomy. I have never seen any permanent bad results follow the removal of part or all of the uvula, if no other tissue was damaged. I have seen several patients in whom the mental effect was bad because of the knowledge than an accident had occurred, but they were unable to name definite symptoms. I have never seen any bad results when the anterior pillars on one or both sides were removed or injured. I believe that bad results do follow, at times, severe injury or removal of the posterior pillars. The symptoms vary, depending on the amount of tissue damaged, and whether one or both sides were involved. The speaking voice is not usually affected, but with a singing voice, the more delicate tones are always altered. This may not be appreciable to the lay ear, but a professional critic will be conscious of a lack of quality and of tone fineness. There is often a sense of constriction and fulness about the throat that annoys the singer, and he loses confidence in his vocal ability.

When the posterior pillars are removed, or severely injured, there are often contractions sufficient to interfere with the normal suction or negative pressure exerted by the palate on the postpharyngeal wall. This allows the nasal pharynx to retain its secretion, which predisposes to infections with the usual complications. These conditions occur too frequently after severe injury or removal of the posterior pillars to be disregarded. I have never seen severe contractions at the base of the tongue, when only the lymphoid tissues in the tonsillar fossae had been removed. In my opinion, these contractions are always due to some muscle injury. Paralysis of the palate with fluid regurgitation through the nose has always been temporary, due to swelling of tissue. It is not a true paralysis. To my knowledge, I have never had a voice injured by a tonsillectomy. I always warn mezzo sopranos and lyric sopranos that they may possibly lose one or two upper tones, but eventually, in two or three months, these tones will return, and the quality of voice will improve. I explain this temporary loss by the fact that the fulcrum action of the glossopharyngeal

muscle over the tonsil in raising the larynx is disturbed, and requires time for compensation.

I have had three cases of infection in the tonsil fossae, all occurring after local anesthesia, when incision through the anterior pillar was necessary for drainage. In a six months' period, when I was using an unusually long needle (1¼ inch), I saw two cervical abscesses, which I had to drain externally. I believe the needle passed through the superior constrictor muscle, planting infection in the soft tissues of the neck. I have shortened the needle about one-half. Wherever and whenever laryngologists discuss any phase of tonsil work, the question of granulation tissue in the fossae comes in for much unnecessary debate. Wound healing takes place in two ways. First, by primary intention. The edges of the wound are approximated, which is impossible in tonsil surgery. Second, by secondary intention or granulation, when wounded edges are not brought together. This is the only possible mode of healing after a tonsillectomy. Coagulated fibrin and blood serum form in the fossae, the dead elements are removed by the phagocytes, and a proliferation of the connective tissue along with the accumulated leukocytes produces the formative, or embryonic tissue. Capillaries spring from the surrounding vessels, and the embryonic tissue changes into granulation tissue, which is gradually replaced by true connective tissue. This is an absolutely normal process of healing in all open wounds. True, granulation tissue may become excessive at times, and require attention to hasten healing, but if left undisturbed, it eventually will be replaced by connective tissue. Granulation tissue should never be confused with lymphoid tissue which is left in the fossae. I believe that the granulation tissue question is a mask behind which an incomplete tonsillectomy is usually hidden.

As to the lingual tonsil, at the angle of the tongue, turning into the fossae after operation: The lingual tonsil is a distinct anatomic entity without migratory ability. I cannot understand by what anatomic acrobatics it could ever sever its connection on the pharyngeal dorsum of the tongue, and transplant itself into the tonsil fossae. Scar tissue might draw it toward the fossae, but its base would still remain attached to the tongue.

Dr. Pratt states: "There is a great question in the minds of most men on the amount of tissue to remove at the base of the tongue at the time of operation." Does he mean the base of the tongue, or the inferior tonsil fossae? True, they are in close proximity, and there is a contiguity of tissue. In my opinion, all the lymphatic tissue in the entire tonsil fossae should be removed at the time of operation, but never any tissue from the base of the tongue, unless there is a distinct indication for doing so.

CARCINOMA OF THE LARYNX AND LARYNGO-PHARYNX TREATED WITH RADIUM

AN ANALYSIS OF THIRTY CASES *

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During the last three years, fifty-three patients with carcinoma of the larynx and laryngopharynx have been diagnosed and treated at the bronchoscopic clinic of the Manhattan Eye, Ear and Throat Hospital. Of these patients thirty have been under continuous observation. The remaining twenty-three, after having been diagnosed as patients with carcinoma of the larynx and laryngopharynx, received from one to two treatments with radium but were then completely lost trace of, so that this report must necessarily deal with but thirty case reports.

The radium dosage was regulated and controlled by Dr. Robinson, who is in charge of the radium at the Manhattan Eye, Ear and Throat Hospital. In all the patients treated, when the radium was applied internally it was done by using the element in 5 to 10 mg. platinum needles. When the radium was applied externally, it was done by the block method.

Most of the patients were far advanced in the disease when first seen. Some had refused operation at the hands of the men in the nose and throat clinics and others were so far advanced that operative measures would have been futile. With but few exceptions, the type of growth was extrinsic, and if operative methods had been practiced a laryngectomy plus type of operation would have been necessary.

Of course, in such advanced types of cases radium treatment was primarily considered to be of low efficiency.

A condensed record of these cases is here submitted.

The following is the outline used in the collection of the data:

Clinic Number
Sex — Name
Age — Habits: Tobacco
Alcohol

Family history
Past history relating to the larynx
Character of first symptom noted, particularly onset of hoarseness, dysphagia, cough or pain
Date of first examination
Physical Examination: Teeth (pyorrhea, bad, fair, good, artificial)
Glands
Pharynx
Larynx—location of growth: intrinsic
extrinsic
anterior
posterior
midportion
Motility of cords
General appearance of growth

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* From the bronchoscopic clinic of the Manhattan Eye, Ear and Throat Clinic.

* Read before the American Laryngological Association, Montreal, Canada, June 5, 1926.

Biopsy report
Method of application of radium
Date of last examination and general description of growth
Condition when last seen or heard from
Cured
Alive
Alive and apparently improving
Alive, relieved by operation
Dead
Remarks

REPORT OF CASES

CASE 1.—A. S., aged 55, a laborer, who used tobacco and alcohol, complained of hoarseness of six months' duration. The family history was negative. July 17, 1923, there was subglottic involvement extending to the valeculla and by way of the glosso-epiglottic fold; it was very hard in consistency. The glands were palpable. Pyorrhea was noted in the teeth, and the patient had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: July 20, 1923, four 10 mg. needles for four hours; April 4, 1924, three 10 mg. needles for three hours. The patient improved for about six months, but died, June 18, 1924.

CASE 2.—R. M., a man, aged 50, a driver, a user of tobacco, with a negative family history, had had some dyspnea for the last month. July 20, 1923, a large mass covering the introitus of the larynx attached to the right aryepiglottic fold was noted, and a large mass infiltrating the right pyriform fossa. Glands were palpable in the right side of the neck; the teeth were poor, many of them decayed. The patient had chronic pharyngitis. The biopsy report, July 20, was fibropolyps; September 14, squamous cell carcinoma.

Radium was applied as follows: September 21, four 10 mg. needles for four hours; October 5, four 10 mg. needles for four hours; December 17, four 10 mg. needles for four hours. There was never any improvement, and the patient died in May, 1924.

CASE 3.—N. H., a man, aged 58, a cook, who used tobacco and alcohol, had a negative family history. Nov. 25, 1923, there was ulceration and infiltration of the laryngeal surface of the epiglottis, with infiltration of the left aryepiglottic fold. Pain was felt on swallowing; there had been no voice changes for the last four months. No glands were palpable. The teeth were good. The patient had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: November 26, four 2 mg. needles in the epiglottis and the aryepiglottic fold for two hours; December 28, two 5 mg. needles for two hours, and Oct. 11, 1924, two 5 mg. needles for two hours.

April 1, 1924, the patient complained of pain in the chest. A roentgenogram showed a mass in the left upper lobe, probably metastasis. It was reported that he died, Aug. 1, 1925.

CASE 4.—G. G., a man, aged 74, who used tobacco and alcohol, had a negative family history. Jan. 7, 1924, there was a mass in the left lateral wall of the pharynx, the aryepiglottic fold and the ventricular band and cord. He had felt pain and difficulty in swallowing for the last two months. Glands were palpable; the teeth had been extracted. He suffered from chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: January 22, three 10 mg. needles for three hours; March 7, five 10 mg. needles for four hours; April 4, block, 1,050 mg. hours, and April 11, block, 1,050 mg. hours. The patient was unimproved, and died in June, 1924.

CASE 5.—H. G., a man, aged 60, a user of tobacco and alcohol, had a negative family history. April 4, 1924, there was a large cauliflower-like mass in the right pyriform fossa extending to the arytenoid. He had had difficulty in swallowing and a slight amount of pain for the last month. Glands were palpable. The teeth were in very bad condition. He suffered from chronic pharyngitis. The biopsy report was squamous cell carcinoma.

Radium was applied as follows: May 29, block, 960 mg. hours; June 6, five 10 mg. needles for three hours; July 11, three 5 mg. needles for six hours, and September 12, block, 2,160 mg. hours. The patient was unimproved and died late in October, 1924.

CASE 6.—W. R., a man, aged 50, a user of alcohol, had a negative family history. May 13, 1924, there was an infiltrating mass in the right side of larynx, involving the cord, the ventricular band, the arytenoid and the aryepiglottic fold. The voice had been hoarse for the last four months, and there was some difficulty in swallowing. Glands were palpable. The teeth were all in bad condition, and the patient had chronic pharyngitis. The biopsy report was squamous cell carcinoma.

Radium was applied as follows: May 16, block, 1,600 mg. hours; June 20, five 5 mg. needles for five hours; July 22, three 10 mg. needles for six hours, and November 14, two 10 mg. needles for six hours. The patient was unimproved, November 14, and was reported dead, July 18, 1925.

CASE 7.—W. S., a man, aged 66, a user of tobacco and alcohol, had a negative family history. Sept. 9, 1924, there was swelling of the left arytenoid, a small area in the left pyriform fossa and the lateral pharyngeal wall. Pain in the left ear, cough, and some difficulty in swallowing had been present for the last three weeks. The glands were very large in the left side of the neck. The teeth had been extracted. The patient had chronic pharyngitis. The biopsy report was epithelioma.

Radium was applied as follows: September 11, block, 1,500 mg. hours; September 21, block, 2,100 mg. hours; October 21, three 10 mg. needles for five hours; November 20, block, 1,500 mg. hours; Jan. 7, 1925, block, 1,050 mg. hours; February 11, block, 3,400 mg. hours; March 18, block, 2,000 mg. hours; April 1, block, 2,000 mg. hours, and June 6, block, 4,000 mg. hours. The last report, March 2, 1926, was that the patient was alive but very weak.

CASE 8.—P. K., a man, aged 58, a user of tobacco, gave in his family history that his father died from cancer of the throat. Jan. 7, 1924, there was a mass in both pyriform fossae, and he had difficulty in swallowing. The glands on both sides of the neck were palpable. The teeth were bad. The patient had chronic pharyngitis. The biopsy report was squamous cell carcinoma.

Radium was applied as follows: Jan. 1, 1924, three 5 mg. needles for three hours; April 3, four 5 mg. needles for four hours. The results were: Feb. 6, 1925, some ulceration of the growth was present. There had not been enlargement in the size of the growth. Jan. 18, 1926, the condition generally was about the same. The patient had not experienced any further difficulty in swallowing, and the general appearance of the growth was the same as Feb. 6, 1925.

CASE 9.—P. R., a man, aged 49, a user of tobacco, had a negative family history. Dec. 4, 1924, the posterior wall of the pharynx and the right arytenoid were infiltrated. He had slight difficulty in swallowing and the glands were just palpable. The teeth were not noted. The biopsy report was carcinoma.

Radium was applied as follows: December 4, block, 3,150 mg. hours; December 5, three 10 mg. needles for three hours. The patient was unimproved, Jan. 16, 1925, and died, April 7.

CASE 10.—M. M., a man, aged 62, a user of tobacco and alcohol, had a negative family history. Feb. 20, 1925, there were general infiltration of the right pyriform fossa and the right arytenoid, some difficulty in swallowing and some hoarseness. The glands were palpable in the right side of the neck. The teeth were not noted. The pharynx was negative. The biopsy report was carcinoma.

Radium was applied as follows: March 11, block, 4,000 mg. hours; April 15, block, 1,000 mg. hours, and June 10, block, 3,200 mg. hours. No improvement was made and the patient died, July 4.

CASE 11.—E. E., a man, aged 49, a user of tobacco and alcohol, had a negative family history. Jan. 21, 1925, there was mass infiltration of the left cord, the

ventricular band, the arytenoid, and the left lateral pharyngeal wall and epiglottis. Hoarseness, difficulty in swallowing and pain were noted. There was a large mass in the left side of the neck. The teeth were in poor condition. He had chronic pharyngitis. The biopsy report was squamous cell carcinoma.

Radium was applied as follows: January 30, block, 4,000 mg. hours; February 6, block, 4,000 mg. hours; March 6, block, 2,000 mg. hours. Gastrostomy was done in June, and the patient died, August 10.

CASE 12.—J. O'M., a man, aged 69, a user of tobacco and alcohol, had a negative family history. Jan. 19, 1925, there were involvement of the left cord and ventricular band and hoarseness. The glands were not palpable. The teeth were bad. The patient had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: June 29, block, 1,600 mg. hours, and July 27, block, 2,700 mg. hours. Thyrotomy was done January 30. The last report was that the patient was in very poor condition, with general involvement of the larynx and laryngopharynx.

CASE 13.—D. S., a man, aged 35, a user of tobacco, had a negative family history. He had had a cyst of the epiglottis removed about six months previously, and there was cystic involvement of the epiglottis and aryepiglottic fold. He had some difficulty in swallowing. The glands were not palpable. The teeth were not noted. He had chronic pharyngitis. The biopsy report was squamous cell epithelioma. The Wassermann reaction was 2 plus.

Radium was applied as follows: June 15, block, 2,940 mg. hours. The last report was that the patient was alive with no further involvement. The condition was about the same as when radium was applied.

CASE 14.—J. S., a man, aged 53, a user of tobacco, had a negative family history. Jan. 31, 1923, the laryngeal surface of the epiglottis and the left arytenoid were infiltrated with a cauliflower-like growth. Hoarseness, cough and difficulty in swallowing were noted. The glands were palpable. The teeth were in bad condition. He had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: February 6, three 10 mg. needles for four hours, and March 9, four 10 mg. needles for four hours. The patient died in October, 1924.

CASE 15.—J. S., a man, aged 74, a letter carrier, who used alcohol and tobacco, had a negative family history. April 4, 1923, there was a growth on the anterior portion of the left cord, about 0.5 cm. in diameter and hoarseness was noted. The glands were not palpable. The teeth were artificial. There was a slight degree of pharyngitis. The biopsy report was basal cell epithelioma.

Radium was applied as follows: April 11, 1923, three 5 mg. needles for two hours. Examination immediately following the biopsy showed that the growth seemed to be totally removed. Subsequent examination showed no recurrence, and the patient appears to have been cured, the last examination having been a few weeks before this article was written. The hoarseness had entirely disappeared.

CASE 16.—F. H., a man, aged 65, a user of alcohol and tobacco, had a negative family history. June 22, 1923, there were a large cauliflower-like mass on the left cord and hoarseness. The glands were palpable. The teeth were all in poor condition, and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: June 29, five 10 mg. needles for four hours, and August 17, three 10 mg. needles for four hours. August 19, it was necessary to do a tracheotomy. The patient died, September 9.

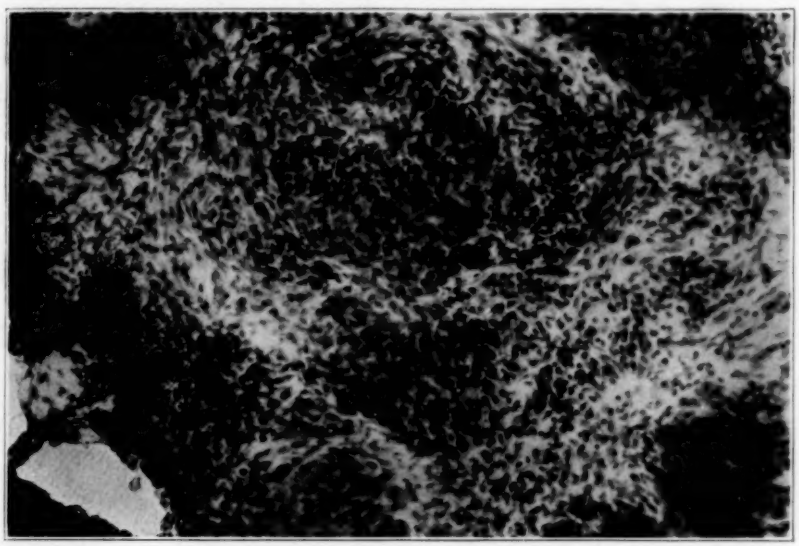
CASE 17.—P. D., a man, aged 54, a user of tobacco and alcohol, had a negative family history. June 29, 1923, there were general infiltration of the upper part of the larynx, with ulceration of the left cord and arytenoid, hoarseness and some difficulty in breathing. Glands were palpable. The teeth were in bad condition, and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: June 29, block, 700 mg. hours; October 29, block, 1,400 mg. hours. There appeared to be some slight improvement for three months, but the patient died from hemorrhage, December 15.

CASE 18.—C. H., a man, aged 64, a user of neither tobacco nor alcohol, had a negative family history. July 13, 1923, there were fixation of the left cord with infiltration and ulceration posteriorly and hoarseness. The glands were not palpable. The teeth were artificial. The pharynx was normal. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: July 20, three 10 mg. needles for four hours. No further treatment was given. The patient died, December 19, from strangulation.

CASE 19.—P. K., a man, aged 60, a user of tobacco and alcohol, had a negative family history. July 23, 1925, there was general infiltration of the introitus of



Basal cell epithelioma in case 15.

the larynx, necessitating immediate tracheotomy. Glands were palpable. The teeth were artificial. He had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: Four block applications, each 4,000 mg. hours. October 20, the patient was still alive but very weak. No further report was obtainable.

CASE 20.—C. R., a woman, aged 40, had a negative family history. Aug. 10, 1925, there were general infiltration of the upper part of the larynx and epiglottis, difficulty in breathing and swallowing, and hoarseness. Glands were palpable. The teeth were in bad condition, and she had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: August 27, block, 4,000 mg. hours; September 25, block, 4,000 mg. hours; October 14, block, 4,000 mg. hours, and November 23, block, 4,000 mg. hours. A tracheotomy was done, December 15. The patient is still alive but in very poor condition.

CASE 21.—D. N., a man, aged 60, a user of neither tobacco nor alcohol, had a negative family history. Aug. 10, 1925, the right cord and right arytenoid were found indurated with an extensive infiltration.

Hoarseness was noted. Glands were not palpable. The teeth were in bad condition and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: August 17, three 10 mg. needles for four hours, and September 20, four 10 mg. needles for four hours. No improvement was made; the patient had laryngectomy done at another hospital.

CASE 22.—C. M., a man, aged 35, who used tobacco but no alcohol had a negative family history. Aug. 12, 1925, the left cord and arytenoid were infiltrated, with ulceration at the posterior end of the cord. Hoarseness and some difficulty in swallowing were noted. Glands were not palpable. The teeth were artificial. He had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: September 2, block, 4,000 mg. hours, and October 5, block, 4,000 mg. hours. Because of the marked and severe reaction, a laryngectomy was done within a week. The patient is still alive.

CASE 23.—C. P., a man, aged 71, who used tobacco but no alcohol had a negative family history. Nov. 2, 1925, there were general fusiform swelling of the left cord and ventricular band and hoarseness. Glands were not palpable. The teeth were in very poor condition and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: July 15, 1925, block, 3,600 mg. hours; August 12, block, 5,040 mg. hours; September 9, block, 5,400 mg. hours; October 22, block, 4,500 mg. hours, and December 6, block, 3,600 mg. hours. The patient died, Jan. 7, 1926.

CASE 24.—C. C., a man, aged 58, a clerk, who used tobacco and alcohol, had a negative family history. Oct. 11, 1925, there were general infiltration of the left side of the larynx and pyriform fossa with several large ulcerating areas, hoarseness, difficulty in swallowing, and pain in the left ear. Glands were palpable. The teeth were in poor condition and he had chronic pharyngtis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: October 20, five 10 mg. needles for four hours. The patient died from toxemia within four days.

CASE 25.—M. H., a woman, aged 52, had a negative family history Oct. 23, 1925, there was a mass, irregular in outline, on the posterior pharyngeal wall extending down in the left pyriform fossa. She had difficulty in swallowing and pain in the left ear. Glands were palpable. The teeth were bad and she had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: November 27, five 10 mg. needles for four hours, and December 10, five 10 mg. needles for four hours. A gastrostomy was done December 19, and the patient died in March, 1926.

CASE 26.—L. M., a man, aged 52, a user of tobacco but no alcohol, had a negative family history. Nov. 15, 1925, there were infiltration and ulceration of the left side of the epiglottis and aryepiglottic fold, difficulty on swallowing and pain in the left ear and face. The glands were not palpable. The teeth were bad and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: December 9, block, 4,000 mg. hours, and December 19, block, 4,000 mg. hours. He reported one month before this article was written that swallowing was becoming more difficult.

CASE 27.—J. I., a man, aged 52, who used tobacco but no alcohol, had a negative family history. Nov. 22, 1925, there were infiltration and ulceration of the right side of the larynx and epiglottis, difficulty in swallowing and hoarseness. Glands were palpable. The teeth were bad and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: December 1, block, 4,000 mg. hours, and January 3, block, 4,000 mg. hours. The patient feels better, can swallow better, and generally appears to be improved. The mass in the larynx and epiglottis does not seem to have increased in size.

CASE 28.—A. B., a man, aged 50, a user of tobacco and alcohol, had a negative family history. April 3, 1924, there were infiltration and ulceration of the epiglottis, difficulty in swallowing and pain in both ears. Glands were palpable. The teeth were bad and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: April 10, two 10 mg. needles for four hours; June 18, block, 900 mg. hours, and July 15, two 10 mg. needles for four hours. March 15, 1926, he reported by letter that he was unimproved.

CASE 29.—J. R., a man, aged 58, a tailor, who used neither tobacco nor alcohol, had a negative family history. May 11, 1925, on the anterior two thirds of both cords there was an irregular whitish growth, that was diagnosed as precancerous, and hoarseness was present. Glands were not palpable. The teeth were artificial. He had chronic pharyngitis. The biopsy report, May 11, 1925, was keratosis; Jan. 12, 1926, squamous cell epithelioma.

Radium was applied as follows: May 19, 1925, block, 2,800 mg. hours; August 18, block, 2,100 mg. hours, and September 25, three 10 mg. needles for two hours. The growth had remained the same up to January, 1926, when a laryngectomy was urged but refused. At present the patient is being treated with high-voltage roentgen ray by Dr. F. C. Wood. There seems to be a decided decrease in the size of the growth since this treatment was started.

CASE 30.—A. R., a man, aged 60, a tailor, who used tobacco but no alcohol, had a negative family history. June 11, 1925, there a large ulcerating mass on the right of the larynx at the site of the cord and ventricle and ventricular band, hoarseness and pain. Glands were palpable. The teeth were bad and he had chronic pharyngitis. The biopsy report was squamous cell epithelioma.

Radium was applied as follows: June 15, block, 2,800 mg. hours. Because of increasing obstruction, the patient consented to a thyrotomy. Considerable necrosis of the cartilage was encountered by the operator, Dr. Faulkner. The patient is alive.

SUMMARY

In the thirty cases studied, twenty-eight were males, two, females. The ages ranged from 35 to 74 years. As to occupation sufficient data could not be obtained to be of any value in this report. The data as to family history were not sufficient to be conclusive. There was one patient (case 8) who recalled that his father died of cancer of the throat. The past history was of negative importance as regards symptoms referable to the larynx. There was one patient who had a cyst of the epiglottis removed six months previously (case 13).

The first symptoms noted by the patients were hoarseness in eighteen; dysphagia in eighteen; pain in 8, and dyspnea in 3. There was positive evidence of enlargement of the glands in twenty-three patients; they were not palpable in seven patients.

The teeth were good in one patient; artificial in nine; bad in eighteen, and not noted in two.

The pharynx was normal in four patients, and twenty-six had chronic pharyngitis.

There were thirty biopsy reports, and of these twenty-nine were squamous cell epithelioma and one basal cell epithelioma (case 15).

The number of applications of radium in each case ranged from one to nine, the average being between two and three. There were forty-nine block and thirty-one needling applications given. The usual happening following the use of radium was that more or less of an edema of the whole growth occurred. This usually subsided within a week or so. This was particularly noted when needles were used. The wound of entrance of the needle occasionally showed as an edematous spot. One patient developed an acute toxemia following the application of the radium and died within four days. Another developed a milder degree of toxemia, on whom a laryngectomy was done. This patient is still alive. The longest period of observation was three years (case 15).

A summary of the present status of the cases is as follows:

(*a*) Cured, one (case 15). This was a basal cell epithelioma and, as stated elsewhere, it was felt that the cure was effected at the time the biopsy was done, although it is very possible that the few cancer elements at the base of the growth were destroyed by the use of radium. Three 5 mg. needles were inserted for two hours into the outer border of the cord. This patient is now 77 years old and his vocal cord shows no evidence of the site of the growth nor any loss of motility. He has a very good voice.

(*b*) Alive, nine (cases 7, 8, 12, 13, 19, 21, 26, 27 and 28). These patients are alive but very weak, and enduring all the tortures and sufferings that only a patient with cancer of the throat has during the last few months of life.

(*c*) Alive and apparently improving, one (case 29). This patient neither smoked nor drank. A diagnosis of a precancerous growth was made over a year previous to the report of a squamous cell epithelioma obtained in January, 1926. Radium was applied as noted, but it seemed that the growth was enlarging, so a laryngectomy was urged but the patient positively refused any operative measures. He was then referred for high voltage roentgen-ray treatment, and after twenty treatments there was a decided decrease in the size of the growth.

(*d*) Alive after tracheotomy, one (case 20).

(*e*) Alive after thyrotomy, one (case 30).

(*f*) Alive after laryngectomy, one (case 21). These require hardly any comment, except to say that the last two patients whose histories were detailed in cases 30 and 21 are alive because of these operations. In case 30 there was considerable sloughing following the application of the radium, and in case 21 a moderate degree of toxemia.

(*g*) Dead, sixteen (cases 1, 2, 3, 4, 5, 6, 9, 10, 11, 14, 16, 17, 18, 23, 24 and 25).

CONCLUSION

The cure of cancer of the throat, in all but exceptional cases, would not seem to be attained through the use of radium.

17 East Thirty-Eighth Street.

Clinical Note

THE AUDIO-CONTROLLER

O. C. SMITH

JACKSONVILLE, ILL.

WITH A PREFACE BY

GEORGE E. SHAMBAUGH, M.D., CHICAGO

Recently, while conducting certain examinations of the children in the Illinois School for the Deaf at Jacksonville, I became interested in an apparatus called an Audio-Controller, which I observed being used in the institution, an apparatus which has been devised and worked out by Col. O. C. Smith, managing officer of the school, and Mr. R. W. Blucke, an electrician. The mechanical as well as the pedagogical principles seemed to be entirely sound. It appeared to me that we had here an apparatus that would assist very materially in the tremendously difficult problem of instructing the deaf. In order that the otologic profession might become acquainted with the device, Colonel Smith, at my request, has prepared the following statement:

After several years of experimentation, and through the use of the radio vacuum tube, it has become possible to revolutionize the method of instruction of deaf students. Not only is this statement true, but other wonderful feats are now a posibility. For instance, many people who have not heard a sound for years are able to hear distinctly, and without any effort on their part, all ordinary conversations transpiring in any room in which they are. With the apparatus as now developed and as described, it is not necessary for just one person at a time to direct his voice into a certain mouthpiece or microphone, but, on the contrary, any and all sounds, or voices in any part of the room, are picked up and made audible to the individual or to any number of individuals.

This has been made a possibility by inventions of myself and Mr. R. W. Blucke. We have been working on and developing the apparatus described here for the past three years.[1]

The apparatus consists of a super-sensitive electrical microphone, which records with precision every sound wave. This microphone, in turn, transforms each and every sound wave into a corresponding electrical wave or impulse which is an exact duplicate of the original sound wave. This primary or original electrical impulse is conducted by means of suitable coupling transformers to the grid element of a three element radio vacuum tube. This vacuum tube is known as the first stage of amplification. The principle, or the action, of the vacuum tube is to reproduce accurately in a highly magnified form the weak electrical impulses applied to the grid element. In other words, the output of this tube is many times greater than the input, and is an exact electrical wave reproduction of the original sound waves.

There are several tubes cascaded to give several more stages of amplification. The final stage represents thousands of times of amplification of the original electrical impulse. The output of the last tube is led to a distributing block.

1. Mr. R. W. Blucke has been closely connected with radio for fifteen years, and I am Managing Officer of the Illinois School for the Deaf.

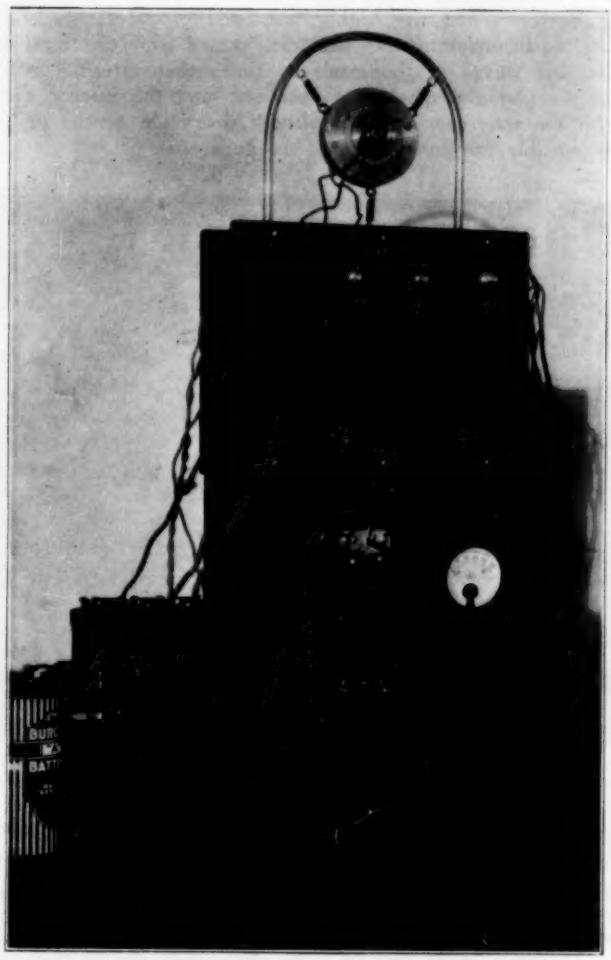


Fig. 1.—Smith audio-controller.

From the distributing block a number of branches or leads are run to an equal number of head phones. These head phones are controlled individually by each listener by means of a carefully worked out piece of apparatus known as a volume controller. Each person can control the volume of sound in their phones so that same is adjusted properly to their ears.

This is necessary, as it has been noted that a volume of sound suited for one person would be entirely unsuited for another, because of the degree of deafness of each. When the highly amplified electrical waves strike the diaphragm of the headphones, they are transformed from electrical waves back again into sound waves of thousands of times their original volume. The closeness of the phone diaphragm to the ear, and the intense amplification, together with the true reproduction without distortion of the original voice, has been responsible for the success of the apparatus.

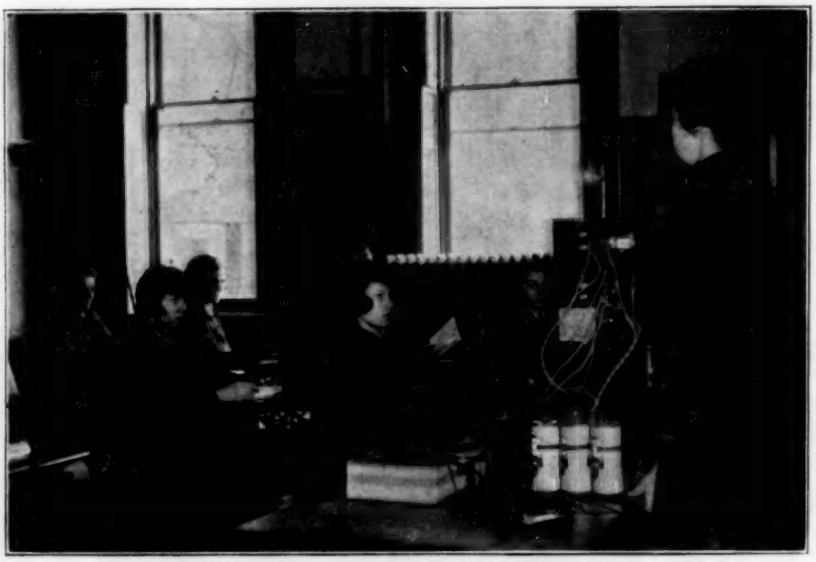


Fig. 2.—The audio-controller in use.

This instrument is designed solely for the purpose of giving hard of hearing children an education through the medium of their hearing, and no claim is laid that the instrument has a curative value.

The use of this instrument has passed the experimental stage, as it has been clearly demonstrated by using it on a class of eight pupils that these children can make twice the progress by the use of the instrument than they can without it. They also have a different conception of life, talk better and read lips much better than other children in the same grades in this school. They seem to hear better, but it is a question as to whether their hearing has been built up or whether it has been educated.

My opinion is that in all cases it is a question of them being able to recognize words, rather than to hear words that they have never heard before.

PROGRESS IN OTOLARYNGOLOGY

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

INTRADURAL COMPLICATIONS OF AURAL AND NASAL ORIGIN

A SURVEY OF RECENT LITERATURE

WELLS P. EAGLETON, M.D.
NEWARK, N. J.

(Continued from page 69)

FRONTAL LOBE ABSCESS

Jessaman [34] states that abscess of the frontal lobe is not of frequent occurrence, the most common causes being infection secondary to frontal sinus suppuration and trauma. The mortality is very high, a large factor being the great difficulty in making an early diagnosis. Persistent headache is probably the most common symptom, and its continuance after the drainage of infected sinuses should be regarded with suspicion. Optic neuritis is rare. The occurrence of convulsions or paralysis in the presence of infection of the accessory nasal sinuses is strong evidence of an intracranial complication, and demands prompt attention.

Jessaman reports a case of persistent right sided headache and obstructed drainage from the right frontal sinus; the anterior part of the right middle turbinate was removed and the anterior cells opened. Improvement followed, but two days later the patient had a convulsion, became rather drowsy, and complained of severe pain in the head. A large opening was made in the posterior wall. The dura was rather dark and appeared under tension. Incision of the dura did not show any *pus beneath it, but the brain was discolored.* An incision through about one-fourth inch of the brain cortex opened an abscess, from which a large amount of dark colored pus was evacuated. This abscess must have been very close to the lateral ventricle. Subsequently there was considerable trouble in keeping drainage, and finally a curved cautery was employed to burn down the granulations from the frontal sinus to the nose.

Arnoldson and Bostroem [35] also report a frontal lobe abscess successfully treated after a second abscess had been drained; it was suddenly manifested by convulsions.

Cowan [36] reports a case of an intrapia-arachnoid abscess. The patient died as the result of a collection of pus in the left lateral ventricle.

34. Jessaman, L. W.: Abscess of the Frontal Lobe Secondary to Suppuration of the Frontal Sinus: with Report of Case, Boston M. & S. J. **192**:739, 1925.

35. Arnoldson, N., and Bostroem, C. G.: Cerebral Abscess of Rhinogenic Origin, Acta oto-laryng. **8**:339, 1925.

36. Cowan, H. Alexander: Intradural Abscess Secondary to Frontal and Ethmoidal Sinusitis, Lancet **2**:648, 1925.

Frequency of Multiplicity.—For some unknown reason the cerebellum is much more frequently the site of several separate foci of suppuration than the large brain.

Many of the supposed separate abscesses on microscopic examination will be found to have a small communication with the larger primary cavity. These abscess prolongations are undoubtedly caused by compression from the dural envelop, as stressed by Fraser,[37] who at postmortem found three abscess cavities in the left cerebellar hemisphere, only one of which had been opened at the operation. The pressure effect was shown by a well marked herniation through the foramen magnum.

Fremel [38] in a contribution on the "Morphology and Growth of Cerebellar Abscess," reports two multiple out of three cases of cerebellar abscess.

The first patient with chronic left sided otorrhea was admitted with fever and headache in the left frontal and parietal region. The dura was found to be exposed over the tegmen tympani. The sinus was thrombotic. Two weeks later puncture of the cerebellum evacuated a large abscess. Death followed at the end of nine days. Necropsy showed a very tense dura with flattened walls, smooth and free from exudate; the left cerebellar hemisphere was enlarged, and a seropurulent exudate of a greenish color was seen in the vicinity of the incision.

The second patient had suffered from left sided ear suppuration, with partial loss of hearing; the otorrhea returned after a cold bath. No vomiting, fever, vertigo or aural tinnitis was present. Radical operation was followed by the formation of a *retropharyngeal abscess.* Meningitic symptoms supervened. A later operation revealed a granulation on the dura behind the labyrinth; puncture yielded discolored pus. Death occurred two hours after operation. Necropsy showed a large opened abscess in the left cerebellar hemisphere, filled with blood clot. The abscess occupied the entire covering of the cerebellar hemisphere, and the encephalitis reached the ependyma of the fourth ventricle. The pus contained *Streptococus pyogenes.* Histologic sections of the cerebellum showed two abscess cavities.

The third patient had chronic bilateral suppurative otitis media. There were attacks of severe headache, especially severe at night, followed by vertigo, nausea and vomiting. Temporary improvement occurred without operation. Necropsy showed three cerebellar abscesses the size of a nut, separated by the cortex.

Comment: The multiplicity of abscesses can perhaps be interpreted as individual processes of a common abscess cavity, especially as the abscesses were separated by the cortex and pia.

Massesi [39] contributes a good anatomic description of an abscess situated in the anterior one third of the cerebellum—the usual position when the infection originates from caries of the petrous pyramid.

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37. Fraser, J. S.: Chronic Middle Ear Suppuration; Cholesteatoma; Latent Labyrinthitis; Cerebellar Abscess; Death, Proc. Scottish Soc. Otol. & Laryngol., Twenty-First Meeting, J. Laryngol. & Otol., no. 9, **40**:598-599 (Sept.) 1925.

38. Fremel, F.: Morphology and Growth of Cerebellar Abscesses; Monatschr. f. Ohrenh., Laryng. Rhinol. **60**:128, 1926.

39. Massesi, B.: Cerebellar Abscess of Otitic Origin, of Unusual Localization, Gior. di chir. med. **5**:164. 1926.

Necropsy on the transverse section of the cerebellum showed that a large portion of the *right* cerebellum was replaced by an enormous cavity filled with pus. The destroyed nervous substance comprised a large part of the right hemisphere and a portion of the vermis.

Diagnosis of Cerebellar Abscess.—(*a*) Indefiniteness of Localizing Symptoms: Gavello,[40] of the University of Turin, observed five otogenic cerebellar abscesses in 1918-1919. Recently he recorded three more cases in which the diagnosis was rendered difficult, or impossible, because of the indefinite symptoms and the serious condition of the patient, which prevented an examination. The site of the abscess was always on the side of the affected ear. He says that from the diagnostic point of view the location of the headache is of slight importance, as it was altogether absent in some patients during certain periods of the disease, and in other cases, was diffusely distributed, and was limited to the nape of the neck only in a few cases. Rigidity of the neck and tenderness on percussion of the bones therefore have no special significance. Vomiting may be present or absent. Changes of the ocular fundi were observed but were not invariably present; neither did spinal puncture furnish an accurate guide. The most regular and noteworthy symptom consists in more or less marked spontaneous nystagmus; which may be produced by pressure on the vestibular nuclei (Deiters' nucleus), as well as from the cortex of the hemispheres. Gavello thinks the nystagmus does not differ from labyrinthine nystagmus as its direction may be toward the diseased, as well as toward the normal ear. When a labyrinthine cause of the nystagmus can be excluded, it is a valuable diagnostic sign of cerebellar affection. The vertigo is parallel with the nystagmus. Disturbances of equilibrium are not dependent on definite positions of the body or the head as distinguished from labyrinthine affections. Associated facial paralysis is directly connected with middle ear disease. The tests of cerebellar localization, such as disturbances of the coordinated muscular function on which modern cerebellar diagnosis is based, could not be carried out by the author on account of the grave condition of the patients.

On the other hand, Nühsmann [41] states that the spontaneous nystagmus of cerebellar abscess is at first to *the same side*, but within a few hours after the destruction of the vestibular end organ, it is to the healthy side, from the unopposed action of the opposite labyrinth. When the head is turned toward the sound side, the patient falls forward instead of backward to the diseased side as in cerebellar abscess. Some time between the eighth to the twenty-eighth day the roof nuclei become

40. Gavello, G.: Cerebellar Abscesses of Otitic Origin, Arch. ital. di chir. **12:** 567, 1925.

41. Nühsmann, T.: The Differential Diagnosis Between Inflammatory Labyrinthitis and Otogenic Cerebellar Disease, Arch. f. Ohrenh- Nasen- u. Kehlkopfh. **43,** no. 4 (July) 1925.

compensated to the altered condition, and with this adjustment the nystagmus, past-pointing, falling, and locomotory disturbances disappear.

(*b*) Conflicting Vestibular Manifestations: Jones [42] contributes a case which shows how misleading the vestibular findings may be when regarded entirely from a localizing standpoint; as associated with an aphasia (perhaps due to arterial thrombosis), they caused an exploration of the temporal lobe, when in reality the abscess was situated in the cerebellum.

The patient had otitis media and mastoiditis. The vestibular findings were: Prompt response to nystagmus, with total absence of vertigo; tendency to conjugate deviation on stimulation of both horizontal canals; the presence of response from left vertical canals on first examination, followed at a later date by absence of response, incoordination of ocular movement, which came into evidence from ear stimulation—the deviation of the left eye inward on douching the right ear, and the deviation of the right ear inward on douching the left ear.

Necropsy revealed in the region of the left cerebral cortex, covered by the bone flap, two small areas of necrosis extending into the subcortical white tissue. (These areas apparently corresponded to the points into which the exploratory needle had entered at the operation.) There was enlargement of both lateral ventricles. There was a large abscess, which occupied practically the entire right cerebellar hemisphere, with a pouting area of pus directly adjacent to the mastoid cavity (making it evident that there had been a direct extension through the dura at this point from the mastoid).

(*c*) Localizing Vestibular Symptoms: That the *induced* vestibular manifestations, however, may be of the greatest localizing value, especially in *acute* lesions of the cerebellum, is shown by Mauthner,[43] who reports a case of hemorrhagic encephalitis and cerebellar abscess after measles and subsequent otitis, with histologic findings.

The patient had a mastoditis as a sequel of measles and acute otitis media. After operative intervention she lost consciousness. A few hours later, the eyeballs were immovable, the extremities rigid (in the sense of Sherrington's decerebration rigidity). *Cold irrigation of the right ear was followed by raising of the right eye and lowering of the left.* Necropsy revealed encephalitic foci in the medulla oblongata, an abscess of the central portion of the right cerebellum and a small thrombotic blood clot in the third ventricle.

Vestibular Tests in Nonsuppurative Lesions.—Wishart [44] summarizes the present status of the diagnostic localizing value of the ear tests, and states that his evidence permits of the following postulates:

1. Tumors of the frontal lobes do not interfere with cochlear or vestibular function.

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42. Jones, Isaac H.: Cerebellar Abscess with an Unusual Complication, Laryngoscope **35**:893-895 (Dec.) 1925.

43. Mauthner, O.: Only Apparently Otogenic Cerebral Complications in Acute Infectious Diseases, Monatschr. f. Ohrenh. **59**:672, 1925.

44. Wishart, D. E. S.: Neuro-otological Examination in Seven Consecutive Verified Cases of Brain Tumor in the Posterior Fossa in Children, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:86-147, 1925.

2. Subtentorial tumors which are completely outside the brain stem and the cerebellum affect homolateral cochlear and vestibular function.

3. Subtentorial tumors which are in the midline and which do not involve cerebellar tissue do not affect cochlear function. They do interfere with vestibular function. The interference tends to be bilaterally symmetrical, and the functions of both vertical canal reflex arcs are affected before those of either horizontal canal.

4. Unilateral intracerebellar tumors do not interfere with cochlear function. They do interfere with vestibular function. The function of the reflex arcs of the vertical canals will both be affected before those of the horizontal canals. The tumor will be found on the side opposite to the vertical canals the function of which is most interfered with.

Fisher,[45] however, would add that vertigo and past-pointing were found to be reliable indications of the condition of posterior fossa contents. A cerebellar lesion, with very few exceptions, should show impaired vertigo and past-pointing. However, the presence of good vertigo and past-pointing is even more strongly indicative of a normal cerebellum. Another feature of cerebellar lesions is the lack of susceptibility of those patients to vestibular stimulation. Nausea, pallor, sweating and vomiting as a result of douching and turning, were practically never encountered in cases of cerebellar disease, and their presence was used almost as a definite indication that the lesion was not cerebellar.

Treatment of Brain Abscess.—The only real advance in the surgical technic of brain abscess is contributed by Cahill,[46] who reports the largest number of successive cures of brain abscess ever recorded. His method may be summarized as (1) attempting to produce protective adhesions of the pia-arachnoid prior to dural incision, and (2) later establishing drainage by a wide-mouthed wire cone sutured into the dural opening.

Cahill says:

> When the mastoid route is used, the dura of the middle fossa is exposed to a diameter of three centimeters. The cavity is packed with dichloramine T, or mercurochrome gauze, for forty-eight to seventy-two hours. Then the dura is opened with a crucial or three-limbed incision, after passing silk sutures through the tip of each of the dural flaps. The operator seeks the abscess with the grooved director or Cushing needle. The anterior portion of the exposed area should be used for exploring the anterior portion, and the posterior area for exploring the posterior portion of the temporal lobe. If the abscess is found, without

45. Fisher, Lewis: The Present Status of Vestibular Tests in Intracranial Conditions, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:148-156, 1925. (Record of 103 suspected cases of brain lesion. Cases selected were verified by operation or necropsy; operative findings.)

46. Cahill, Harry P.: Twelve Cases of Cerebral and Cerebellar Abscesses Drained by the Mosher Wire Gauze Cone, Tr. Am. Otol. Soc., Fifty-Eighth Annual Meeting, pt. I, **17**:42-66, 1925.

permitting the grooved director to shift to the slightest degree, the drain with its plunger in place is guided along the grooved director into the abscess.

The cone should be held in place by granulations after the first week. If it becomes blocked, it may be gently curetted out. In the third week the skin and dural sutures may be removed and the brain permitted to expel the drain.

Errecart [47] drains by means of a glass tube with perforations 5 mm. in size, projecting from the surgical wound in such a way that the gap in the dura mater remains well separated. The tube is not expelled by the pulsating movements of the brain, and there is a protection against the occurrence of cerebral hernia.

Beck [48] describes Neumann's method for exposure of the floor of the anterior portion of the middle fossa.

The patient, with a history of chronic left sided middle ear suppuration, was admitted in an unconscious condition, with symptoms of typical meningitis. Spinal puncture revealed a purulent fluid with innumerable polymorphonuclear cells. The bone at the base of the skull, which appeared softened and discolored, was removed. The exposed dura of the middle cranial fossa showed thick fibrinopurulent deposits and in one place appeared necrotic. Exploratory puncture of the temporal lobe yielded pus. Because the purulent deposits on the dura extended into the anterior portion of the middle fossa, the temporal muscle was detached in its fascia from the zygomatic arch and turned upward. It was thus possible to resect a large portion of the squama and the zygomatic process; this was followed by the removal of the bone of the middle cranial fossa to within 1 cm. of the foramen ovale. The meningeal symptoms subsided after eight days.

Results of Surgical Treatment of Cerebellar Abscess.—That marked advance in the surgical treatment of cerebellar abscess has been made during the past few years is shown by the lowered mortality in the Halle clinic.[41] From 1911 to 1920, of twenty-six cases of brain abscess, eight were cerebellar, with only one survival; while from 1921 to 1924, of twenty-one abscesses, ten were cerebellar, with seven survivals.

After-Treatment of Brain Abscess; Rest and Selection of Type of Work.—Fraser,[49] in reporting a case, emphasizes the importance of a prolonged period of enforced rest after evacuation of an abscess. Failure in such after-care has been followed by death in numerous instances.

The patient did very well for six days, and was allowed to get up, but at the end of half an hour he complained of headache and vomiting, and his temperature rose. Postmortem examination revealed an abscess of the right temporal lobe which had ruptured into the lateral ventricle; there were red softening and hemorrhages round the abscess, basal meningitis and corking of the cerebellum into the foramen magnum.

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47. Errecart: Drainage of Otitic Cerebral Abscess, Rev. españ. y Americ. de laringol., 1925, p. 25; l'oto-rhino-laryng. internat. **11**:467, 1925.

48. Beck, O.: Extensive Brain Abscess—Labyrinthitis—Operation—Recovery, Wien. klin. Wchnschr. 1926, no. 11, p. 514.

49. Fraser, J. S.: Acute Middle Ear Suppuration (right); Extradural Abscess; Temporal Lobe Abscess; Ventricular Meningitis; Death, Proc. Scottish Soc. of Otol. and Laryngol., Twenty-first Meeting, J. Laryngol. & Otol. **40**:593-595, no. 9 (Sept.) 1925.

Dr. Ewart Martin, in the discussion of Fraser's case, raised the question as to how long after recovery patients should be allowed to return to work.

In a patient operated on by him, recurrence took place six months after drainage of the abscess, and it was again opened. Shortly afterward the patient had a fit of the jacksonian type. Three months afterward a decompression operation was performed and a blood clot removed from the area of the abscess. The patient worked as a gardener which entailed a certain amount of stooping, and developed further attacks of jacksonian epilepsy. He then acted as a valet, without any stooping, with no recurrence of epilepsy.

MENINGITIS

Surgical Anatomy.—Future progress in the treatment of meningitis must largely depend on a better understanding of the anatomy and physiology of the pia-arachnoid. Not sufficient importance has been attached to its minute anatomy since Key and Retzius' [50] work in 1875 until Locke and Naffziger [51] in 1924 and Karlefors [52] in 1925 published their important papers.

Boss [53] says that since the publication of Bárány's complex (which was explained through dropsy of the cisterna pontis lateralis), these spaces do not seem to have acquired the importance to which they are entitled in otologic surgery, especially in the therapy of otogenic meningitis. He investigated the topographic relations of these cisterns to one another and to the petrous pyramid by the injection of a methylene blue and agar mixture.

He summarizes as follows: 1. The cisterna magna (or cerebellomedullaris) is bounded anteriorly by the pons and the fourth ventricle, above by the lower vermis of the cerebellum, behind by the falx cerebelli. Ventrally, it extends to the medulla oblongata, where it communicates with the anterior basal cisterns. 2. The cisterna pontis media contains the basilar artery. 3. The cisterna pontis lateralis is the only cisterna in relation with the petrous pyramid. It is directly applied to the anterior portion of the mesial pyramidal surface, and practical importance is attached to the fact that the labyrinth, the meatus acusticus internus and the cochlear aqueduct open into it. It is traversed by the glossopharyngeal, vagus, facial, acoustic, trigeminal and abducens

50. Key, Axel, von; and Retzius, G.: Studien in der Anatomie des Nervensystems und des Bindegewebes.

51. Locke, Charles Edward, Jr., and Naffziger, Howard C.: The Cerebral Subarachnoid System, Arch. Neurol. & Psychiat. **12**:411-418 (Oct.) 1924.

52. Karlefors, J.: The Meningeal Spaces of the Cerebellum; Communications of the Fourth Ventricle with Subarachnoid Spaces and the Cochlear Aqueduct in Man, Acta oto-laryng., 1925, suppl. 4.

53. Boss, L.: Topography of the Arachnoid Cisterns, Zentralbl. f. Chir. 1926, no. 9, p. 542.

nerves. In rare cases, it passes with the pontile cerebellar peduncles into a larger subarachnoid space of the horizontal cerebellar sulcus.

Types of Otitic and Rhinitic Meningitis.—Wharry [54] states that Milligan has differentiated three types of meningitis of otitic origin: (1) meningitis benigna (or serous); (2) meningitis semimaligna (in which pus cells are present in the cerebrospinal fluid); (3) meningitis maligna (in which both pus and organisms are present). Of patients with type 3 cases, only 2 or 3 per cent have been known to recover irrespective of any treatment. From what Wharry has seen, the majority of the few patients with meningitis maligna who recover, appear to have staphylococcal cases.

Wharry reports the case of a patient with staphylococcic meningitis of otitic origin, who was operated on and recovered after transthecal irrigation. A large fistula was found in the lateral semicircular canal. The infection had evidently passed through the labyrinth and lamina cribrosa into the internal auditory meatus and thus to the basal meninges. The lamina cribrosa was removed to allow as free drainage as possible of the basal cisterns along the route of infection (Scott-West method). The right internal ear was irrigated through the spinal canal with Locke's solution at body temperature.

Pathology.—The microscopic studies of Turner and Reynolds [25] show how infective thrombophlebitis gives rise to leptomeningitis—the usual terminal process. The leptomeninges showed diffuse purulent meningitis with septic thrombosis of branches of the middle meningeal and superficial middle cerebral veins. In some instances, the wall of the vein had broken down and pus had escaped into the pial meshes.

Pathologic Origin of Cortical Symptoms.—Přecechtěl [55] describes three cases, which may be summarized as follows: In all, the earliest symptoms of meningitis were localized cortical signs; in one, irritative signs and convulsions followed by paralysis; in the other two, paralysis without any preceding stage of irritation.

He says that cortical signs have a different significance in the two classes of cases; when appearing suddenly in the course of an acute otitis media, they suggest the onset of a "hood" meningitis, and the prognosis is accordingly unfavorable.

Rhinorrhea and Its Relation to Meningitis.—That a large proportion of patients with rhinorrhea die from a complicating meningitis, and those who live become blind, was demonstrated by St. Claire Thompson a quarter of a century ago.

The etiology is obscure, but Beck, in discussion, said he had seen two specimens dissected by Chiari which showed deficiency in the cribri-

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54. Wharry, H. M.: Three Cases of Middle Ear Disease with Intracranial Complications, Brit. M. J. **1**:85, 1926.

55. Přecechtěl, A.: Disturbances of the Motor Cortex in Otogenic Meningitis, Zentralbl. f. Hals- Nasen- u. Ohrenh. **7**:797, 1925; abst. J. Laryng. & Otol. **41**:259 (April) 1926.

form plate, with a fistula that had never closed. I have seen a rhinorrhea following a fracture of the skull, with death from meningitis a year later, which at postmortem showed that the watery discharge from the nose originated from a chronic traumatic pial cyst of the brain which drained through the frontal sinus. Up to the present time, no progress has been made in its treatment, although the suggestion of placing a strip of fascia lata was suggested to the patient.

However, Stein,[56] in reporting a case, states that the discharge almost ceased for two weeks after the administration of benzyl benzonate solution, 20 drops, three times a day, and after three weeks it entirely disappeared and has not recurred in five months. Atropine internally had no effect, and alcohol injection of the left Meckel's ganglion was performed without result. At the time of the report, the patient had a feeling of wellbeing with no increase of headaches.

Kuemmel[57] reports a case of a girl, aged 18, who had been suffering from atrophic rhinitis of two years' duration, with frontal headache and a mucopurulent nasal discharge. An injection of her own blood was made under the mucosa of the right lower turbinal and in the right side of the septum. Some days later, another injection of about 3 cc. of blood was made under the left lower turbinal and in the left side of the septum. Both underwent complete absorption. The third injection of about 5 cc. of blood under the mucosa of the lower turbinal and the septums of both sides led to the formation of an abscess in the left frontal brain, hemolytic streptococcic meningitis and death.

Meningitis following Gradenigo Syndrome.—The statistics of Sears[58] demonstrate that death from meningitis supervenes in more than 15 per cent of the cases of Gradenigo syndrome.

Gradenigo[59] of the University of Naples says that while numerous theories have been advanced as to the origin of his syndrome, he is of the opinion that the abducens paralysis is only of extradural origin. The infection may spread from the small peripheral pneumatic cells of the petrous bone and by a lymphatic route, or between the carotid artery and the wall of the cavernous sinus. The lymphatic path was demonstrated by his pupil Papele.

Engelhardt[60] observed that in one instance abducens paresis did not develop until the onset of terminal purulent meningitis, although at

56. Stein, Otto J.: Cerebrospinal Rhinorrhea, Chicago. Laryng. & Otol. Soc. Proc., April 6, 1925; Ann. Otol. Rhin. & Laryng. **34**:1269-1271 (Dec.) 1925.

57. Kuemmel, W.: Two Unusual Cases of Rhinogenic Meningitis, Acta otolaryng. **8**:209, 1925.

58. Sears, W. Hardin: Otogenic Paralysis of the Abducens, with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion, Tr. Am. Laryng. Rhinol. & Otol. Soc., 1925.

59. Gradenigo, G.: The Syndrome of the Apex of the Petrous Bone, Rev. otoneurol. ophth. **2**:158, 1925.

60. Engelhardt, G.: Abducens Paralysis in Extradural Abscess at the Apex of the Petrous Bone, After Acute Middle Ear Suppuration; Death from Meningitis, Ztschr. f. Hals,- Nasen- u. Ohrenh. **11**:194, 1925.

necropsy there were changes at the petrous apex. He thinks that the value of the symptom of abducens paresis is variable, as it is often absent in extradural abscess at the apex of the petrous bone until the meninges are affected. On the other hand, it may be present in middle ear suppurations which heal without major intervention.

Ulrich [61] reports a case in which the entrance of infection to the meninges was demonstrated only on microscopic examination. The postmortem examination showed that the original focus was extradural and affected the tip of the petrous bone. The gasserian ganglion was invaded and materially damaged by suppuration, and the sixth nerve was reduced in size to a mere thread.

There was an acute exacerbation of a chronic middle ear suppuration, calling for paracentesis and mastoid operation. The symptoms subsided with the exception of the abducens paralysis. Two months later there came on suddenly giddiness, vomiting and diffuse headache, followed by stupor, stiffness of the neck, Kernig's sign and exaggerated reflexes; with *Streptococcus mucosus* in the cerebrospinal fluid.

Recurrence of Meningitis After Apparent Cure.—Experimental evidence [62] demonstrates that the micro-organism of suppurative meningitis may remain dormant—hidden as it were—in the subarachnoid prolongations of the cranial nerves as they leave the skull, and then suddenly take on renewed activity.

Lannois and Jacod [63] report a case of "Otogenous Suppurative Meningitis with Remissions," with purulent but sterile cultures.

The meningitis lasted altogether three months, with two remissions, one of short duration before operative intervention, during which the patient resumed his work, another, of about one month, after a mastoid evacuation. During the remissions, the cerebrospinal fluid was clear.

Treatment of Meningitis.—Slowly the treatment of septic meningitis is resolving itself into (1) early diagnosis while the process is still limited to an area of the pia-arachnoid adjacent to the primary focus of infection, (2) the drainage of this area; to which I [64] have added the necessity of (3) putting the parts at rest by ligature of the internal carotid artery, and (4) attempting to increase the immunity of the cerebrospinal fluid by repeated blood transfusions from an immunized donor, associated with massive lumbar punctures.[65]

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61. Ulrich, K., Zurich: Otogenous Abducens Paralysis, Ztschr. f. Hals-Nasen- u. Ohrenh. **9**:403 (Jan.) 1925.

62. Weed, L. H.; Wegeforth, P.; Ayer, J. B., and Felton, L. D.: Experimental Meningitis, Monog. Rockefeller Inst. Med. Res., March 25, 1920, no. 12, pp. 57-112.

63. Lannois and Jacod: Rev. de laryngol., Bordeaux **45**:1 (Jan. 15) 1924.

64. Eagleton, W. P.: The Surgical Treatment of Meningitis (Third Communication), J. A. M. A. **83**:1900-1906 (Dec. 13) 1924.

65. Eagleton, W. P.: Meningitis of Otitic Origin (Fourth Communication), presented to the Section of Otolaryngology, A. M. A., 1926.

Goerke [66] would open wide the dura mater of both cranial fossae, because he believes it is only in this way that the paths of infection can be followed and the primary foci of intracranial infection be removed.

He does not think that the question when to open the labyrinth is as yet definitely settled. Total loss of labyrinthine function does not always call for opening, as it may be caused by a serous or a past fibrous inflammation. On the other hand, in the presence of a functioning labyrinth it should be opened when the clinical picture is suggestive of a circumscribed labyrinthitis. With meningitic symptoms, even a functioning labyrinth should be sacrificed. After the radical removal of the source of infection, the next step is the eradication of the invading organisms and the neutralization of their toxins by medicinal therapy. He states that the majority of the many recommended remedies, such as vucin, optochin, eucupin, protargol, collargol, electrargol, argochrome, dispargen, and others, have been found to be inefficient. He has some faith in trypaflavin and methenamine. He does not recommend intradural introduction of methenamine by means of spinal puncture as it reaches the infectious focus only in very weak concentrations, but thinks that the best results are obtained by intravenous introduction. Spinal puncture is an important adjuvant, as it leads to the lowering of the brain pressure, which is followed, in its turn, by the production of a new bactericidal cerebral fluid, and by an improved absorption through the pachionian granulations.

The last and most important step is drainage of the subarachnoid space. A permanent drainage is not secured by the mere opening of the dura mater, or by the introduction of a cannula. The large cisterna cerebromedullaris is not accessible from the ear, whereas the cisterna pontis lateralis with its ramifications is within easy reach. After wide exposure of both cranial fossae, the dura is raised with a spatula from the posterior surface of the petrous.

Nühsmann [67] reports forty cases of purulent meningitis seen during the last four years in the Halle University ear clinic. He thinks the principal treatment is the elimination of the primary pus focus in the mastoid process and repeated spinal punctures. He regards an increase of the polymorphonuclear leukocytes in the cerebrospinal fluid as generally indicative of an extension in the meningitis and as an unfavorable sign even with a temporary improvement of the clinical symptoms. On the other hand, a diminution of the polymorphonuclear leukocytes in the puncture fluid or an increase of the lymphocytes is a favorable prognosis even with a discouraging clinical picture.

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66. Goerke, M.: Treatment of Otogenic Meningitis, Zentralbl. f. d. ges. Chir. u. i. Grenzgeb. **33**:512, 1925.

67. Nühsmann, T.: Treatment of Otogenic Purulent Meningitis, Arch. f. Ohrenh.- u. Kehlkopf. 1925, 113, p. 16.

He thinks that the injection of vucin solution into the spinal space is not only useless but often harmful on account of the injury to the spinal meninges and abscess formation in the skin.

Kepes' [68] case of cured otitic meningitis is important because it shows the more favorable prognosis of meningitis when the source of infection of the meninges can be determined, such as in sinus thrombosis, and points the way to the routine method of operation, namely, by following the path of infection into the meninges, whether from the sinus, a small vein, the labyrinth or a brain abscess.

A boy had acute middle ear suppuration followed by symptoms of sinus phlebitis. The turbid cerebrospinal fluid contained *Streptococcus longus*. Operation revealed no thrombus, and consequently Kepes thinks that the initial symptoms were presumably caused by Koerner's osteophlebitis. There was a brain abscess in the middle cranial fossa.

Treatment by Septicemin.—Mavens [69] reports two cases of otogenic meningitis cured by septicemin after operation, one from a chronic otitis, the other from acute labyrinthitis. In the first case, with bidaily spinal punctures, he injected septicemin intraspinally (an iodine-tropin compound) first 1, then 2 ampules of 4 cc., until the cerebrospinal fluid, which had contained staphylococci and streptococci, was free from bacteria. Septicemin was also administered intravenously. The second patient was cured by intravenous injections of septicemin and local irrigation of the operative wound.

Results of Treatment.—Of fifty patients with meningitis treated in the Breslau ear clinic during the last five years, sixteen were cured; nearly one third of the cases. These favorable results are referred by Hinsberg [70] to (1) early recognition of the meningitis and removal of the pus focus in the ear or in the nasal sinuses; (2) the subsequent treatment by large doses of methenamine (6 to 10 Gm. daily, in part intravenously, in part by mouth). Hemorrhagic cystitis almost invariably supervenes, sometimes within a few days, often delayed until over 200 Gm. of the remedy has been administered. He is in favor of suboccipital puncture, because it does not involve the danger of a flow of cerebrospinal fluid like incision of the cisterna pontica.

Vaccine and Serum Treatment of Meningitis.—In one of the clinics in France, cases of brain abscess (following evacuation) are treated as a routine with an autogenous vaccine to prevent, if possible, the secondary staphylococcic infection, which so frequently occurs. Treatment of the

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68. Kepes, P.: Pathology and Symptomatology of Otogenic Meningitis; Cured, Zentralbl. f. Hals,- Nasen- u. Ohrenh. **8**:54 (Oct. 25) 1925.

69. Mavens: Curability of Septic Purulent Meningitis of Otitic Origin, Ann. d. mal. de l'oreille, du larynx **44**:28, 1925.

70. Hinsberg: Results of Treatment of Purulent Meningitis, Klin. Wchnschr. **3**:1648, 1924.

meningitis by all forms of vaccine and serum therapy alone has been uniformly unsuccessful, as is shown by Barajas' [71] case.

A woman, aged 50, had a right sided ear suppuration of twenty-four hours' duration, and some pain in the corresponding side of the head. Simple antiphlogistic and antiseptic treatment was instituted, with reactivation of the general defensive forces through 0.5 cc. of the antipyogenic polyvalent Bruschetti vaccine. A comatose state suddenly supervened on the following day, deepening into absolute unconsciousness, with convulsions, conjugate deviation of the eyes and a rise of temperature. The patient when seen by Barajas presented the picture of diffuse purulent meningitis. Operation was refused. Spinal puncture yielded a purulent fluid with abundant polymorphonuclear cells and a large quantity of streptococci. The patient died a few hours later.

However, vaccine treatment combined with operation, removal of the source of infection, undoubtedly has had a favorable influence in a few reported cases of meningitis; but, as in the case of Ferretti,[72] they are open to the suspicion that the patient has suffered from meningismus only—without micro-organisms in either the spinal or cerebral fluids.

A man with acute otitis media, after seven days, presented the signs of labyrinthine involvement, with vertigo, headache and tremors. On the tenth day, he had retraction of the head and Kernig's sign. An extensive mastoid operation was performed. Spinal puncture four times daily, with evacuation of large quantities of turbid fluid (30 to 40 cc.). An autogenous vaccine was prepared and injected intramuscularly. Immediate improvement followed, and the patient was discharged in good condition at the end of a month. A recurrence took place three weeks later, but a complete cure was obtained by spinal puncture and irrigation of the mastoid cavity with surgical solution of chlorinated soda (Dakin's solution).

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71. Barajas, y de Vilches, J. M.: Hyper-acute Otogenic Meningitis, Siglo méd. **76**:345, 1925.

72. Ferretti, C.: Leptomeningitis Due to Acute Otitis, Arch. ital. di otol. **36**: 628, 1925.

Abstracts from Current Literature

Ear

PATHOLOGICAL STUDIES OF THE TEMPORAL BONE. JOSEF FISCHER, Monatschr. f. Ohrenh. **60**:137 (Feb.) 1926.

Exostoses of inflammatory origin have incorrectly been classified with osteomas; they belong preferably to the category of acute and chronic periostitis. Osteomas occur in the external auditory canal, in the antrum, the mastoid process and the region of the lateral semicircular canal. A specimen is pictured with several circumscribed osteomas on the posterior surface of the pyramidal bone.

Many tumors of the temporal bone reported as sarcomas are in reality endotheliomas. This error has exaggerated the number of actual sarcomas. The malignancy of the tumor is recognized in the microscopic picture. Metastases are very rare.

The point of origin of carcinoma is usually the skin of the external canal or the auricula. In a few, the condition was found originating on the plastic flap of a mastoid on which a radical operation had been performed. The irritant factor in the etiology of a carcinoma is generally the chronic otorrhea. Epithelioma of the external canal develops rapidly and extensively but seldom metastasizes. Destruction of the neighboring bony structures is very marked due to pressure necrosis of the tumor, aided by the toxicity of the cancer cells.

GLOWACKI, Detroit.

CONTRIBUTION TO THE CLINIC AND TREATMENT OF TUBERCULOSIS OF THE MIDDLE EAR. W. SPOHN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:580 (March 20) 1926.

Spohn differentiates between tuberculous middle ear suppurations, which show no specific general symptoms and those in which general tuberculosis is present. He describes seven cases in detail.

Case 1.—The patient, aged 55, suffered from tuberculosis of both lungs, mastoiditis and labyrinthitis. A radical operation was performed. Cheesy masses were found in the mastoid, and the semicircular canal was eroded. Death followed two months later, from the tuberculosis.

Case 2.—The patient, aged 46, suffered from tuberculosis of both lungs, and insisted on an operation on account of severe headache and pain in the mastoid process. Antrotomy was performed. The patient died two months later.

Case 3.—A man, aged 28, was admitted May 22, 1923. He had beeen under constant care since the war on account of lung trouble, and had been operated on in another clinic December, 1922, on account of dizziness and headache. This time, no operation was performed; the wound cavity was cleansed. The patient died January, 1924, of tuberculosis.

Case 4.—A man, aged 26, was admitted Aug. 7, 1923. In 1921 he had suffered from pneumonia and apical catarrh. At times, since February, 1923, the patient was confined to his bed, because of tuberculosis of the right apex. In August, he suffered from mastoiditis with swelling behind the ear. The radical opera-

tion revealed breaking down of the whole mastoid. In September, the facial nerve became paralyzed. Curetting showed that the facial nerve was freely exposed. After a few weeks the patient died from tuberculosis of the lung.

Case 5.—A man, aged 38, was admitted Oct. 28, 1924. In July he had been stabbed repeatedly in the chest. Since October he had been in a lung hospital. Otitis media occurred which was followed by mastoiditis in five weeks; the paracentesis did not improve the patient's condition. November 1, the mastoid antrum was opened; November 25, he returned to the lung hospital. Jan. 26, 1925, slow healing was reported. The patient was discharged from the hospital Feb. 3, 1925.

Case 6.—A man, aged 32, was admitted Nov. 3, 1924. In 1912 he had had a hemorrhage from the lung; in 1924, violent hemoptysis had occurred. At the time of admission the patient suffered from bilateral open tuberculosis and a chronic middle ear suppuration, probably of a tuberculosis nature. Within three weeks the discharge increased, and a radical operation was performed. Three weeks later meningitis developed, with fatal results. Necropsy showed miliary tuberculosis of both lungs, cavities in the left lower lobe, tuberculosis of the kidney and epididymis, miliary tuberculosis of the liver and spleen and grayish white nodules on the vessels of the sylvian fossa.

Case 7.—The patient, aged 9½ months, was admitted Dec. 12, 1924. Both ears were discharging since October. A facial paralysis of the left side developed in the beginning of December. The mastoid region was swollen and reddened; the canal was swollen, and blood and pus were found in the depth. A radical operation showed an extensive destruction of the posterior wall of the meatus. The dura was intact. The Pirquet and percutaneous reactions were strongly positive. The spinal fluid showed a moderate lymphocytosis, coagulum but no microbes. The child died fourteen days later of meningitis.

Indications for operation vary; the subjects with acute symptoms of mastoiditis and great pain which progressed, gave indication for operation. Other patients were operated on because of increased secretion, pronounced headache, repeated exacerbations and facial paralysis. A postoperative labyrinthian exacerbation which occurred in the presence of evident tuberculosis was not operated on. The therapy, with exception of a single case, was surgical. The prognosis for a complete cure was doubtful in all cases. The question arises whether conservative treatment would have been preferable, and whether operations were at all justified. With some patients the operation seemed to be without any promise of success, on account of the general condition. In others, the object of the operation, the cure of the annoying discharge, was not obtained. The patients probably could have lived without surgical interference. Even when tuberculosis had attacked the whole mastoid process with erosion of the semicircular canal, a quick progress into the endocranial cavity was not to be expected. These cases confirm the idea of conservative treatment. If the condition of a tuberculous ear is aggravated by an acute secondary infection, or by an exacerbation to an extent that a retrogression of the symptom cannot be expected, respectively, surgical interference must be considered. A surgical interference was necessary in progressing acute cases of mastoiditis with unbearable pain, because then the patient can be treated later for the general condition in proper surroundings. No case of tuberculosis is entirely hopeless (Brieger, Uffenorde) therefore, an attempt to help is justified in many instances. (The reviewer is not in the habit of expressing an opinion about any article which he reviews. In this instance,

however, the author does not mention the nature of the anesthesia employed, whether it was local or general, and, if general, what kind of an anesthesia was used).

PELVIC REFLEXES OF THE EYES IN MAN AND THEIR SIGNIFICANCE FOR THE WEAK TURNING TEST OF THE VESTIBULAR APPARATUS. KARL GRAHE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:613 (March 20) 1926.

A working man, aged 29, who had become deaf three years previously through meningitis, came to the university polyclinic on account of violent pain in the frontal region. Diseased nasal accessory cavities were found. When the patient was turned while standing, according to Grahe, nystagmus could plainly be palpated and was also visible through Frenzel's spectacles. When the patient stood on the turning chair, and when he was turned so that no change of the position of the pelvis to the body took place, no nystagmus could be felt or be seen through Frenzel's spectacles. When the patient was sitting in the turning chair, the same phenomenon took place. When the patient was placed in a chair and when the legs were abducted and adducted while the pelvis was slightly turned and while the upper parts of the body and the head remained immovable, irregular twitchings of the eyes could be noticed through Frenzel's spectacles, especially when the legs were abducted. Several deafmute pupils were then examined. The pelvic reflexes were found in only one of three children with an unexcitable vestibular apparatus. Stenvers has described a pelvic reflex of the head and eyes. In a patient whose labyrinths responded to irritation, Stenvers saw that turning of the pelvis produced a lasting turning of the head to the same side and deviation of the eyes in the same sense. He considers this phenomenon the position reflex in the sense of Magnus. In this case the movements of the eyes (not nystagmus) appeared only in connection with the turning of the head, whereas in Grahe's patient it could be observed singly. The missing labyrinthian irritability in Grahe's patient also proves that this pelvic reflex has no connection with the labyrinth. Goldstein has made similar observations in patients with supracerebellar affection and normal labyrinths.

In pelvic reflexes of the eyes, the same condition seems to prevail as in nystagmus caused by sensory irritations. In a patient of Grahe's, whose labyrinth had been operated on years ago and who was operated on again on account of cholesteatomatous masses in the internal meatus, nystagmus and past pointing were elicited when the wound cavity was douched with 5 cc. of water at 80.6 F. The patient had been readmitted on account of meningeal symptoms. After the meningeal symptoms had disappeared, the reaction could no longer be shown. Griessmann produced irregularities of nystagmus by placing hot and cold cloths on the neck. Borries claims that reflex nystagmus is only an expression of a generally increased reflex irritation. Pelvic reflexes may simulate an excitability of the vestibular apparatus, under certain circumstances. Grahe supposes that through such a combination of neck and pelvic reflexes of the eyes, the observation of Cemach and Kestenbaum can be explained. These authors could palpate nystagmus in deafmutes whose labyrinths were not excitable, when they were turned in the dark. This fact induced Cemach to deny an irritation of the vestibularis when the subject was subjected to weak turning. Grahe contradicted Cemach. In doubtful cases, it is advisable, when the patient is subjected to weak turning, to place him on the turning chair or the turning footstool in such a manner that the feet do not touch the floor. The observer stands behind the patient, presses the patient

firmly against himself by placing the elbows on the sides and in front of the shoulders and allows his index and middle fingers to rest lightly on the closed eyelids. When the patient is turned, he does not turn his body or neck. The nystagmus can be controlled by palpation.

AMBERG, Detroit.

Pharynx

PRIMARY SYPHILIS OF THE LINGUAL TONSIL. K. NEUMARK, Monatschr. f. Ohrenh. **60**:118 (Feb.) 1926.

Gerber states that the primary sore of syphilis occurring about the mouth is found most frequently on the lips, tonsils and tongue, respectively. The tongue is usually involved on its tip or lateral edges, and rarely on the posterior two-thirds. Review of the literature of the past ninety years discloses only five cases of primary syphilis on the lingual tonsil.

Neumark adds his case to the collection and discusses the differential diagnosis of a lesion at this site between tuberculosis, carcinoma, sarcoma, Vincent's angina, gumma and chancre.

GLOWACKI, Detroit.

Nose

OPTIC NEURITIS IN RELATION TO SINUSITIS. Am. J. Ophth. **9**:361-364 (May) 1926.

At the meeting of the Sections on Ophthalmology and Laryngology of the Royal Society of Medicine at London, Jan. 8, 1926, optic neuritis in relation to sinusitis was considered.

Mr. Affleck Greeves presented a middle-aged woman, in whom retrobulbar neuritis of six months' duration was cured after the sphenoid and ethmoid cells had been explored and evacuated. The culture gave practically pure influenza bacillus. Sir St. Clair Thomson, who did the exploration, confirmed the clinical details.

Dr. A. Logan Turner was able to give a report on the subject of visual disturbances in relation to affections of the nasal cavities and posterior sinuses. This work was done by a committee from the Scottish Society of Otology and Laryngology, and the Scottish Ophthalmological Club and was first formed in March, 1924. Dr. Logan Turner based his remarks on a summary of twenty-eight cases studied by the Edinburgh group. Cases in which the diagnosis was doubtful and instances of syphilis, Leber's atrophy, tumor and tobacco amblyopia were excluded. All the cases in the series belong to the retrobulbar type of optic neuritis, in which the vision, usually of one eye, was fairly rapidly impaired, but showed a tendency to recover spontaneously, and this was the end-result in most of the cases examined. On the rhinologic side the cases had two striking features: (*a*) Almost entire absence of subjective nasal symptoms; (*b*) complete absence of those signs which the rhinologist regarded as evidence of suppurative sinus disease.

For the purpose of discussion, the cases were grouped as follows:

In group A, three (possibly four) cases were regarded by the neurologist as disseminated sclerosis. In this group the vision had returned almost to normal when the patients were last examined.

Group B: In five in this class, resection of the nasal septum was advised to relieve the crowding in the middle turbinal and olfactory region. In two this was performed, the three other patients declining it. In all five the vision returned approximately to normal.

In group C were six cases, in which the posterior sinuses were opened. One of them belonged to group A, as it was disseminated sclerosis. In three of the patients operated on, the sinuses were healthy, as microscopically proved. The vision in two of these normal cases was unaltered by treatment, there being in both a high degree of defect. In the third, though the sinus explored was normal, vision improved from fingers at 1 meter to 6/36. In the other operative cases, no sinus disease was detected and vision afterward improved from hand movements to 6/36 and 6/9 respectively.

In group D were three cases of septic teeth which were extracted, and thereafter vision in two subjects improved to 6/6 and 6/18.

In the last group E, were ten cases in which no nasal interference was carried out. In four the vision improved to 6/6, in one to 6/9, in two to 6/12 and in one to 6/18 when last tested.

Summarizing, Logan Turner said that in none of the twenty-five cases was there any clinical evidence of suppurative sinus disease, and in the six cases in which the posterior sinuses were opened, no pus or secretion was found. Of the six cases in which the posterior sinuses were opened, they were found to be healthy in three. Of the remainder, the lining membrane in one showed slight catarrhal changes and in the other two there was gross evidence of congestion of the mucosa.

Logan Turner felt that in estimating the beneficial effect on the vision of surgical interference in nasal or posterior sinus cavities, regard must be had to the fact that in retrobulbar neuritis there was a tendency toward spontaneous cure, and operation might be performed at a time when the vision was naturally tending toward restoration.

At the same meeting of the Royal Society of Medicine, Mr. E. D. D. Davis said he had studied the notes on seventy-six patients with retrobulbar neuritis who had been referred to him for examination of the nose. Those patients were kept under observation for long periods and were followed up. In thirty, the cause for the optic condition was not discovered, but nasal disease could be excluded. Disseminated sclerosis was present in fourteen, syphilis in nine, septic teeth in seven, and in only seven was there manifest sinus suppuration. Of the cases in which there was nasal sinus suppuration, operation on three patients produced a dramatically rapid improvement in vision; optic atrophy in the other two prevented such a result. Suppuration of the antrum was practically always associated with involvement, also of the frontal and ethmoid cells except when the antral suppuration was due to diseased teeth. In the numerous dental cases which his work enabled him to see, retrobulbar neuritis had never been recorded. Davis felt that careful study of charts of visual fields presented by nasal cases did not provide any aid to diagnosis, nor had he seen retrobulbar neuritis occurring from acute nasal catarrh. If a patient had an increasing retrobulbar neuritis for which no other cause could be discovered, and for which there was reason to suspect sinus disease, he considered it justifiable to remove the middle turbinal and explore the ethmoidal and sphenoidal cells.

H. H. Vail, Cincinnati.

Etiology of Atresia of the Posterior Choana. H. Volowitsch, Monatschr. f. Ohrenh. **60**:113 (Feb.) 1926.

In twenty years the author had only two cases of complete atresia of one posterior choana. The patient here reported, a girl, aged 16 years, since 1918

complained of a fetid breath and rightsided "catarrh" of the nose. She was not aware of the fact that this nasal passage was completely obstructed. Anterior rhinoscopy disclosed a large, fairly firm rhinolith in the right middle meatus, and bony atresia of the choana on this side. Comparison of the right and left sides of the skull on a roentgenogram showed a small maxillary sinus on the right, with thickening of all its walls as well as of the floor of the right nasal cavity.

Many investigators dispense with the consideration of the etiology of choanal atresia by regarding the latter as an intra-uterine anomaly. Denker and Zarniko hesitate to say that in every instance the condition is congenital in nature, believing that atresia may result from some inflammatory process in the nose. This patient showed no scars in the nose and the mucosa was about normal in appearance. The author attributes the thickenings of the bony walls of the nose to a recent inflammation induced by the presence of the rhinolith. Whether the bony atresia of the choana formed in conjunction with the hyperostosis of the lateral nasal wall and floor, Volowitsch does not say.

PHLEBITIS OF SINUS CAVERNOSUS. HANS BRUNNER, Monatschr. f. Ohrenh. **1**:2, 1926.

Detailed macroscopic and microscopic study of the pathology of affections of the sinus cavernosus as a complication of otitis media disclosed the unusually high frequency of this complication. Of twenty-two consecutive ear cases coming to necropsy, twelve had an involvement of the sinus cavernosus. Brunner's findings (54 per cent) are supported by Sternberg, the *prosecteur* of the Vienna Polyclinic. The highest percentage heretofore reported was 16 per cent (Uta).

Other intracranial complications associated with cavernous sinus phlebitis resulting from otitis media were, thrombosis of sigmoidal sinus, two cases; meningitis, seven, and three with meningitis and thrombosis of the sigmoid sinus. In two instances, therefore, no meningeal symptoms were present, the infection extending as far as the cavernous sinus without meningeal involvement.

The route of extension to the cavernous sinus is (1) through the sinus petrosus; (2) retrograde transportation of thrombi from the sinus sigmoideus; and (3) through the veins of the plexus caroticus. The last-mentioned route is important in the causation of cavernous sinus trouble, especially when the middle ear suppuration ulcerates through the eustachian tube into the peri-carotic structures. In those cases in which the cavernous sinus phlebitis is accompanied by meningitis, the mode of infection of the sinus is either through the venae cerebrales inferiores, or the carotic venous plexus, or through caries of the apex of the pyramidal bone.

In the cases studied, abnormal eye findings were very infrequent.

CHOANAL POLYP. KARL MENZEL, Monatschr. f. Ohrenh. **1**:56, 1926.

A heretofore unobserved sign of a nasal polyp extending into the epipharynx is mentioned. Anterior rhinoscopy alone permits the diagnosis of a choanal polyp if on speaking and swallowing the polypous mass, as seen anteriorly, possesses mobility. The explanation lies in the mechanical influence the soft palate and epipharynx exert during the act of deglutition and speech on a mass extending from the nasal cavity posteriorly. GLOWACKI, Detroit.

Miscellaneous

SEASICKNESS. HANS ABELS, Monatschr. f. Ohrenh. **60**:158 (Feb.) 1926.

The subjective sensation of seasickness is due, first, to the afferent impulses arising peripherally from the unaccustomed swaying of the body; second, due to the strangeness of the impulses arising in the labyrinth itself, their great numbers and varied types, all resulting passively (not actively through body actions that are willed).

The upright position of the body receives more afferent impulses and their perception is all the more sensitive. The sensory "regulation" centers are less impressible with the body prone and the eyes closed.

Seasickness is overcome by the self-adjustment of the "regulation" centers to the new and unaccustomed sensations, and the proper coordination of the reactions resulting from the afferent impulses.

ETIOLOGY OF MYRINGITIS BULLOSA. E. V. ULLMANN, Monatschr. f. Ohrenh. **1**:41, 1926.

A very interesting study leads the author to identify myringitis bullosa as a distinct disease, an acute inflammation due to a filterable virus. It resembles herpes in that an inflammatory reaction (keratitis) is induced in rabbits by eroding and inoculating the cornea with the serum obtained from a bulla on the tympanic membrane of the patient. The serum from a bullous myringitis occurring as a feature of acute otitis media, on the other hand, produces no corneal reaction whatever. This coincides with the observations of Doerr (*Zentralbl. f. Dermatol.*, 1925), who demonstrated the immunity of the rabbit cornea to streptococci, staphylococci and pneumococci.

When the myringitis-keratitis has subsided, it has produced an immunity which resists a second corneal inoculation. The same is true of herpes. However, that myringitis bullosa is not identical with herpes was demonstrated in the following manner. Herpes virus was inoculated into corneas that passed through a myringitis-keratitis and that later proved refractive to a second inoculation; a violent keratitis ensued.

The myringitis virus and herpes virus produce certain similar reactions—experimental keratitis and immunity against reinfection—yet are not identical diseases because herpes virus can infect a cornea immune to myringitis virus.

GLOWACKI, Detroit.

OTOGENOUS ABSCESS OF THE SPINAL CORD. M. NONNE, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:574 (March 20) 1926.

Nonne's patient, a child, aged 11 years, had suffered from diphtheria when 7 years old. When 9 years of age, he fell during gymnastic exercises and suffered from a slight concussion of the brain. A few weeks previous to admission the child became ill. The attending physician made the diagnosis of grip. On the second day the right ear began to discharge. The discharge ceased on the fourth day, and the patient got up after being sick for one week. Soon after the child suffered from headache, dizziness and nausea. A radical mastoid operation was done and a thrombus was removed from the sinus sigmoidus and transversus. The next day meningeal symptoms appeared. The spinal fluid showed increased pressure (230 mm.); the Pandy reaction +; globulin reaction (phase I), +; Weichbrodt reaction. Four days later the child complained of numbness in the legs and the next day the legs were paralyzed.

The periosteum and tendon reflexes were absent, also all abdominal reflexes. There was complete anesthesia from the toes, reaching to a line three fingers below the mamillas. Above this zone there was a zone of hypesthesia, two fingers wide. The spinal fluid was clear, not yellow, Pandy reaction (phase I) + +, the Weichbrodt reaction showed traces, cells about 100: 3. Three days later the cell count was 150: 3, mostly lymphocytes. The next day the spinal fluid was yellow and flowed slowly; the Pandy reaction was + + + +; phase I, + + + +; Weichbrodt reaction, + + + +; cells about 80: 3; Quenkenstedt reaction, +. The spinal fluid showed no microbes. The blood culture showed *Streptococcus hemolyticus*. A metastasis in the spinal cord was thought of. While suppurative bronchitis, purulent cystitis, prostate suppuration and gonorrhea have been mentioned as etiologic factors, ear suppurations have not. The possibility of a metastatic osteomyelitis with penetration of the suppuration into the dura was considered, because the xanthochromia in connection with the pronounced globulin reaction, in spite of the lymphocytosis, pointed to a compression. An extradural suppuration may lead, off and on, to a compression of the spinal cord. Such a pachymeningitis externa purulenta can follow an injury, it may accompany tuberculosis, it may be a metastasis of diplococcus bronchitis and it can follow phlebitis with subsequent staphylococcus infection and also furunculosis (staphylococcia). Professor Sudeck performed the laminectomy. The spinal cord was very soft; the dura was intact. There were no extradural changes. An incision in the posterior median line liberated a milky, creamlike fluid. *Streptoccocus hemolyticus* in pure culture was present. The child died of heart failure on the ninth day after the operation. Necropsy showed a suppurative inflammation of the spinal cord from the fourth to the sixth dorsal vertebra, and meningitis.

A Case in Which a Cerebellar Abscess Had Been Diagnosed, Healed by Puncture of the Cerebellum and Incision of the Dura of the Cerebellum. H. Burger, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:598 (March 20) 1926.

Sept. 29, 1925, a man, aged 24, was admitted to Burger's clinic. He suffered from headache and general malaise, his character was changed and he was of a melancholic disposition. January, 1925, he had undergone a radical operation on the left side. The wound had not healed entirely but he had discontinued treatment. Burger found a radical mastoid cavity which was well epidermized in the antrum and aditus. The tympanic cavity was red and moist, and contained some fetid secretion but no granulations and no denuded bone. The left ear was entirely deaf; the right ear was hard of hearing. The pulse, eye grounds and temperature were normal. The patient stood with the feet spread apart. When looking to the left, he had difficulty in standing upright, but there was no trouble when he looked to the right. The Romberg test, even with spread feet, produced pronounced falling to the left and backward. The position of the head did not influence the direction of falling. Spontaneous past pointing to the left was present in the left shoulder joint. There was no past pointing of the right arm, and no past pointing in a vertical direction. Spontaneous nystagmus was not present. The right ear acted strongly on calorization; the left ear did not react to short, cold irritation according to Kobrak, but to hot, short irritation. Cold calorization, according to Bárány, created normal nystagmus after three minutes. With each nystagmus the inclination to fall remained without change to the left and

backward. The neurologic examination by Prof. B. Brouwer showed a doubtful left sided facial paresis, and the symptoms of the eighth nerve before described, but otherwise no change of the cerebral nerves, no disturbance of mobility and sensibility and no signs of meningitis. The following cerebellar symptoms were noted: plain ataxia in the left arm, left sided dysdiadokokinesia, cerebellar asynergia when the body was bent backward, slight but plain ataxia in the left leg, cerebellar catalepsy of Babinski on the left side (only at the first and not at the second examination). All these symptoms, in connection with the disturbances of the gait, and the position of the body, with spontaneous past pointing and the direction of falling which was not influenced by a changed position of the head, also the retained excitability of the left static labyrinth, led to the diagnosis of left sided affection of the cerebellum. On account of the chronic middle ear suppuration on the left side, the diagnosis of a cerebellar abscess on the left side was made. October 4, the cavity of the radical operation was reopened, and the dura of both cranial fossas was exposed. This appeared dirty gray. Granulations or an exudate were not found. Inward from the lateral sinus, several punctures were made with a negative result. When the dura between the labyrinth and sinus was incised, no fluid appeared. In spite of this unsatisfactory finding at the operation, the patient's condition had changed entirely the next day. He was in good humor, and his appetite and faculty for sleep had returned. This improvement continued, and four days after the operation, all cerebellar symptoms had disappeared, namely, past pointing, the inclination to fall, the ataxia, the dysdiadokokinesia. November 6, the cavity of the radical operation was dry. It cannot be assumed that decompression caused the disappearance of the symptoms. There was no symptom of increased intracranial pressure before the operation. The pulse and the fundus of the eye were normal; the sensorium was free and headache was absent. During the operation no prolapse of the brain appeared and no cerebral fluid was evacuated. The condition of the lateral cisterna of the pons and of the saccus endolymphaticus was clear, and meningitis serosa could also be excluded. Lumbar puncture was not made.

SIGMATISM OBSERVATIONS. EMIL FROESCHELS, Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:607 (March 20) 1926.

Among 900 children, Froeschels found 314 who could not enunciate S, C, or Z properly. Thirty-one were not thoroughly examined, and these are excluded from this report. Among others examined were 179 interdental, thirteen addental, seven nasal and sixty-seven lateral sigmatic subjects, whereas seventeen could not produce a sound resembling "S," but they replaced it by D, T, H or Sch (Sh). Of 179 interdental sigmatic subjects, only thirty-eight enounced "S" fairly well interdentally.

Almost 80 per cent showed complicating speech defects, so that they must be placed in the class of multiple interdentality. Complications with other speech disturbances could be found frequently. Nineteen times associative aphasia (stuttering), fourteen times gammacism and kappacism, eight times rhotacism, three times aggramatism, three times open, twice closed and once mixed nasonnement were noticed. Fifty interdental sigmatic subjects also suffered from other disturbances of speech. Thirty-one belonged to the group of stammerers in which group, as is known, these subjects are placed. Stammering is a defect of articulation, which is sometimes caused by anatomic

abnormalities of the speech organ, most frequently, however, by a defective function of articulation. In forty-five cases a retarded speech development, and in eighty-eight cases a normal speech development occurred. In forty-six cases data could not be obtained.

In twenty-three families familial speech disturbances were found. In two instances it occurred in the mother; in seven families, it occurred three times in brothers or sisters of the mother; this disturbance occurred four times in brothers or sisters of the father, and three times cousins of the father stuttered or stammered. Three patients were left-handed. The frequency of multiple interdentality is of interest. It must be attributed to a defective function of the retractors of the tongue, especially of the musculi styloglossi. It is apparent that not the sigmatism but the interdentality must be treated. As "S" is one of the most difficult sounds the treatment should begin with other interdentals.

Addental sigmatism was observed thirteen times. The position of the jaws and teeth of these patients was normal. Among nineteen children who replaced "S" by other consonants, twice a slight prognathism and once a high palate were found. Sixty-seven children belonged to the group of lateral sigmatism. Colorization, according to Gruetzner, and a hearing tube were used, in order to find the places at which the air emanated from the mouth. The author stresses the importance of using a hearing tube besides colorization, as the latter succeeded only forty times. Of complicated speech defects, gammacisms was observed three times in this group; rhotacism once; interdental D and T, twice; "Sch" (Sh) was formed laterally twice, and rhinolalia aperta was present once. Only three of the sixty-seven patients suffered from associative aphasia (stuttering). The lateral tooth arc is supposed to be of great influence in the development of lateral sigmatism. Froeschels is of the opinion that, in many cases, the tooth arc is the consequence of lateral sigmatism and not its cause. He observed four cases of protrusion of some lateral teeth just at the point at which articulation took place. Sometimes only the tooth, which is attacked by the tongue, and not the alveolar process, is bent outward. In view of the fact, observed repeatedly, that the air does not escape in the region of the arc, frequently not even on the same side, it is necessary to revise the hypothesis of the importance of the predisposing factor of anomalies of the jaw and the position of the teeth concerning the lateral sigmatism.

AMBERG, Detroit.

Society Transactions

JOINT MEETING OF THE SECTION ON OTOLOGY AND LARYNGOLOGY OF THE NEW YORK ACADEMY OF MEDICINE, AND THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLOGY AND LARYNGOLOGY

May 19, 1926

FIELDING O. LEWIS, M.D., *in the Chair*

THE MANAGEMENT OF ACUTE SUPPURATIVE OTITIS MEDIA IN THE HOME. Presented by DR. FRANCIS R. PACKARD.

In order to stimulate discussion, and in the attempt to secure some unity of method in this condition, Dr. Packard spoke on this subject. It was admitted by all that prompt and adequate incision of the tympanic membrane is advisable whenever there is distinct evidence of middle ear abscess. The following home treatment was outlined:

1. Early myringotomy.

2. Gentle instillation of cleansing solution and suction of ear secretion, using a glass pipet.

3. Drying of the ear following irrigation.

4. Most any sterile solution will do for cleansing, such as boric acid, physiologic sodium chloride solution, weak phenol (carbolic acid), etc.

5. The use of heat for pain when the mastoid is not involved.

6. The use of cold when there is impending mastoiditis.

7. Secondary myringotomy if necessary.

DISCUSSION

DR. L. M. HURD gives mimeographed instructions to his patients with acute otitis. It is, he feels, important to apply heat and facilitate drainage by frequent cleansing. He accomplishes this by means of constant thermolite application applied through a hole in a piece of cardboard so arranged that the face is protected from the light rays. The secretions are wiped away as they appear.

DR. J. J. KING said it is his practice to irrigate with boric acid solution by means of a rubber bulb.

DR. A. P. VOISLAWSKY irrigates only when drainage is profuse.

DR. GEORGE COATES condemned the common practice of cotton drainage wicks in the canal. In his opinion, such practices tend to hinder free drainage. He uses no cotton. If it is used, it should be changed every half hour. Attention to the nose and throat is most important. For this purpose mild silver protein, silvol, neosilvol, or camphor menthol solutions can be used.

DR. E. V. HUBBARD asked if any members had any experience with the Alpine lamp as a therapeutic measure.

DR. MCFERSON favors the use of some hydrogen peroxide to prevent sealing of the myringotomy opening.

Dr. A. Randall stated that leeching has been abandoned on account of the large opening now made. If properly and early performed, leeching can be of great value. Atropine has considerable local analgesic value (1:200 grains against the tympanum). Aminol is very valuable in controlling pain. Phenol glycerin solutions are beneficial on account of the analgesic and hydroscopic action. In his experience, heat has uniformly given better results than cold. Banding of the ears is important.

Dr. N. P. Stauffer was of the opinion that cold applications tended to limit the bacterial growth, and in this manner exert a more beneficial action than heat.

Dr. Packard, too, felt that when mastoiditis threatened, cold gives better results than heat.

A Contribution to Our Knowledge of the Sphenopalatine Ganglion. Presented by Dr. George Fetterolf, Dr. E. H. Campbell (by invitation) and Dr. K. M. Houser (by invitation). Delivered by Dr. Campbell.

Dr. Campbell first covered the bibliographic history of the sphenopalatine ganglion syndrome and its treatment. Eight patients received alcoholic injection of the ganglion via the posterior palatine canal. These patients had been carefully selected and the diagnosis established by means of a preliminary procaine injection. All were given total or partial relief. The use of ganglion injections for local surgical anesthesia for intranasal work was favorably reported on, and a number of cases were presented.

Dr. Campbell had dissected the ganglion and canal into fifteen specimens. Each of these specimens had been injected with 1 cc. of 70 per cent alcohol stained with methylene blue. In his dissections he found that:

1. The thickness of the palatal tissue over the posterior palatine canal varied from 0.5 to 1.1 cm.

2. In some specimens the canal was large enough to permit lateral motion of the needle. In others, the needle fitted snugly. In the former the ganglion was not equally stained, sometimes the outer and sometimes the inner being more deeply stained, depending on whether the needle had passed along the outer or inner wall of the canal.

3. In nine of the fifteen specimens the needle struck the bone from 3.7 to 4.9 cm. from the palatal surface. This bone was always the anterolateral part of the under surface of the sphenoid sinus. In two cases the needle point would slip off of this surface and penetrate the sphenomaxillary fissure.

4. The sphenopalatine foramen was found usually just above the posterior end of the middle turbinate. In some cases the turbinate extended beyond the ganglion; in others the ganglion was placed superior to the middle turbinate.

5. In nine cases the needle could be passed beyond the sphenoid bone. In eight of these cases the needle was found just internal to the maxillary nerve as it emerged from the foramen rotundum. In the other, it was 0.2 cm. external to this foramen.

6. If the needle was inserted an average distance of 3.96 cm., it was found to reach the level of the vidian nerve. Injection at this level always injected the vidian nerve.

7. In two cases defects of the bony wall of the posterior palatine canal made it possible for the needle to enter the inferior nasal meatus.

8. In all but one, the stain was found to have penetrated the ganglion. When injection was made at a distance of 3.5 cm., the maxillary nerve was not

stained. At 4 cm. the stain was found on the under surface of the orbital tissues. It came no nearer than 0.75 cm. to the optic or ophthalmic nerves. At 4.5 cm. the maxillary nerve was deeply stained and just touched the external portion of the ganglion. All specimens showed some stain in the zygomatic fossa, it having gained this region through the pterygomaxillary fissure.

DISCUSSION

Dr. S. Ruskin was delighted to find that his work had been checked in Philadelphia. Originally, the idea of injecting the ganglion was from Silberman, a dental surgeon, who used this approach to the maxillary nerve. One should be impressed with the dangers of this injection. Not more than 10 to 12 drops of alcohol should be used. Phenol combined with alcohol is to be preferred. The patient should be told that the face may be numb. He has had one case of trophic ulceration and one of temporary abducens paralysis. The optimum distance for injection is 3.5 cm. He has relieved about forty different complaints by this injection. As yet, we know comparatively little of the wide, nervous ramifications and connections of Meckel's ganglion. It is considered quite likely that constant irritation of one of the ganglions of the vegetative nervous system may give rise to vagotonia. This ganglion is the one most often exposed to unnatural conditions.

Dr. Jones spoke on the negative side of ganglion injection. One asthmatic patient was made worse by the injection. He cautioned against undue enthusiasm. When used for nasal operations, the bleeding is more severe than when cocaine is used. Local applications, in his opinion, are preferable.

Foreign Body Accidents During Tonsillectomy. Presented by Dr. Gabriel Tucker.

Dr. Tucker presented a series of eleven cases of aspiration of foreign bodies during tonsillectomy. The objects were mouth gag lamp, piece of tonsil grasping forcep, wire fragment from tonsil snare, tooth crown, deciduous teeth, portion of nasal rasp, gauze sponge, and a cotton swab stick. The diagnosis, symptomatology and prophylaxis of these conditions was considered.

DISCUSSION

Dr. H. H. Forbes said that we are fortunate in having a school of bronchoscopists, who can handle these cases when they arise. He, too, feels with Dr. Tucker that blood stream infection probably explains more lung abscesses than direct aspiration.

Dr. Fowler said that in the last five years, in a clinic treating about ten patients daily, they have had but one inhalation case. In this patient a piece of tonsils lodged in the trachea, which was removed by suction.

Dr. E. V. Hubbard once had a tonsil knife break during operation, but he was able to remove the fragment before it was aspirated.